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THE SCIENCE AND RISK ASSESSMENT BEHIND EPA'S PROPOSED REVISIONS TO THE PARTICULATE MATTER AIR QUALITY STANDARDS

HEARING

BEFORE THE

COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS UNITED STATES SENATE

ONE HUNDRED NINTH CONGRESS

SECOND SESSION

July 19, 2006

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ONE HUNDRED NINTH CONGRESS
SECOND SESSION

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C O N T E N T S

THE SCIENCE AND RISK ASSESSMENT BEHIND EPA'S PROPOSED REVISIONS TO THE PARTICULATE MATTER AIR QUALITY STANDARDS

Wednesday, July 19, 2006

U.S. SENATE,
COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS,
Washington, DC.

The committee met, pursuant to notice, at 9 o'clock a.m. in room 628, Dirksen Senate Office Building, Hon. James Inhofe (chairman of the committee) presiding.

Present: Senators Inhofe, Voinovich, Chafee, DeMint, Isakson, Jeffords, Boxer, Carper, Clinton, and Lautenberg.

Senator INHOFE. Consistent with our policy of starting on time, we will call this meeting to order.

OPENING STATEMENT OF HON. JAMES M. INHOFE, U.S. SENATOR FROM THE STATE OF OKLAHOMA

I would like to tell our members, since we have quite a number of people here and of course we have the WRDA bill on the floor, which I have to manage so I am not going to be able to stay very long, we are going to try to keep everyone to about 3 minutes. I think we put a notice out if we could encourage both sides to do that, and then anyone who arrives after the conclusion of our opening statements would have to just submit the statements for the record, if that is acceptable.

I am managing the WRDA bill, along with my colleague Senator Jeffords, on the floor so I am going to have to go down and prepare for that and won't be able to stay here very long. I am going to ask that Senator Voinovich, who is kind of the master of all air issues anyway, would handle chairing this committee.

Last week, the Air Subcommittee examined the impacts of tightening particulate matter standards on our Nation. Although EPA failed to analyze the regulatory impact on the Nation, these impacts will be enormous. I was particularly struck by the testimony of Harry Alford, president of the National Black Chamber of Commerce, who testified that the greatest health threat to minorities is access to health care and a tightened standard which would threaten the paychecks that cure that threat.

Today, we are examining the science underlying the particulate matter review. The estimated risk today is less than it was estimated in 1997 under Carol Browner when the current standard was set. So while I feel EPA's proposal to tighten the daily stand-

ard to 35 micrograms is overly stringent, I am pleased EPA proposed to retain the existing annual standard.

The rationale to tighten this standard is weak. The EPA cherry-picked what studies it was relying on, downplaying many key studies that shed light on the health effect of particulate matter, some of which are listed on this chart which is difficult even for me to read this close. But anyway, these are the studies.

It also cherry-picked what information it provided to the Clean Air Science Advisory Committee, an important document that seriously skewed the review. A lot of times, CASAC is not given their assigned task. CASAC, the Clean Air Scientific Advisory Committee, is one of a set of some 21 scientists that are supposed to be giving advice and we are supposed to be listening to that advice. They were never told by the EPA that the estimated risk from PM exposure is now considered lower than the risk level estimated during the last review.

EPA's process for this review is also radically different from every previous scientific assessment, calling into question the credibility of the entire review. CASAC is supposed to review relevant science and the public is supposed to provide input. This time, EPA had a cutoff date of April, 2002 which meant that CASAC's assessment does not include almost 4 1/2 years of new studies. Only after I asked EPA to collect the newer studies did it do so. Now, it plans to issue a final rule without an opportunity for public review of how it is assessing those studies.

Worse, the General Accounting Office report being released today shows EPA has failed to follow the National Academy of Sciences recommendations to examine the health effect associated with different kinds of particulates and to incorporate the range of particulate toxicity assumptions into its uncertainty analysis. If we don't know the types of particles causing the health effects, we can't really accurately assess those.

The system that is in place today only considers the size of the particulate matter. I think we all know that the health effects of a molecule of asbestos is much more dangerous than would be a molecule of dust. These things should be considered.

Senator JEFFORDS.

[The prepared statement of Senator Inhofe follows:]

STATEMENT OF HON. JAMES M. INHOFE, U.S. SENATOR FROM THE STATE OF OKLAHOMA

Last week, the Air Subcommittee examined the impacts of tightening particulate matter standards on our nation. Although EPA failed to analyze the regulatory impact on the Nation, these impacts will be enormous. I was particularly struck by the testimony of Harry Alford, President of the National Black Chamber of Commerce, who testified that the greatest health threat to minorities is access to health care and a tightened standard would threaten the paychecks that cure that threat.

Today, we are examining the science underlying the particulate matter review. The estimated risk today is less than what was estimated in 1997 under Carol Browner when the current standard was set. So while I feel EPA's proposal to tighten the daily standard to 35 micrograms is overly stringent, I am pleased EPA proposed to retain the existing annual standard.

The rationale to tighten the standard is weak. EPA cherry-picked what studies it relied on, downplaying many key studies that shed light on the health effect of PM, some of which are listed on this chart.

It also cherry-picked what information it provided to the Clean Air Science Advisory Committee in important documents, seriously skewing the review. For instance,

CASAC was never told by EPA that the estimated risks from PM exposure is now considered lower than the risk level estimated during the last review.

EPA's process for this review is also radically different from every previous scientific assessment, calling into question the credibility of the entire review. CASAC is supposed to review relevant science and the public is supposed to provide input. This time, EPA had a cut-off date of April 2002, which meant CASAC's assessment doesn't include almost 4 1/2 years of new studies. Only after I asked EPA to collect the newer studies did it do so, and now it plans to issue the final rule without an opportunity for public review of how it is assessing those studies.

Worse, as the General Accountability Office report being released today shows, EPA has failed to follow the National Academy of Science's recommendations to examine the health effect associated with different kinds of particles and to incorporate a range of particle toxicity assumptions into its uncertainty analyses. If we don't know what types of particle cause health effects, we may well spend billions of dollars on pollution controls while doing little to improve health.

Thank you.

Senator JEFFORDS. Mr. Chairman, thank you.

Senator INHOFE. Let me just repeat what we said earlier to our new arrivals here. We are going to try to adhere to 3 minutes in opening statements, and then cutoff statements after those who are present have concluded.

Senator JEFFORDS.

OPENING STATEMENT OF HON. JAMES M. JEFFORDS, U.S. SENATOR FROM THE STATE OF VERMONT

Senator JEFFORDS. Mr. Chairman, thank you for having this hearing on the EPA's proposed standards for particulate matter. There is perhaps no more important environmental standard than the national ambient air quality standard for particulate matter. Tens of thousands of Americans die prematurely each year from particulate matter.

The NAAQS are the cornerstone of the entire Clean Air Act. Their fundamental purpose is to tell us when the air is safe to breathe. For more than 30 years, these standards have been set solely on health considerations, using the latest scientific evidence. Congress specifically chose not to allow consideration of cost when setting these standards.

Those who call for a cost-benefit analysis would have us set a standard that fails to protect the health of all Americans. We cannot afford the human costs of such an approach.

Consider this, if the cost-benefit analysis was used to set the NAAQS and the compliance costs of the standards are estimated at $1 billion, the EPA would be required to set a standard that would allow up to $1 billion worth of people to die from air pollution. So just how do we measure $1 billion in human life. Setting the NAAQS in that way was unconscionable. Telling people their air is safe to breathe when it is actually not safe is unacceptable public policy.

We need to revise the existing standards set in 1997. Although the EPA itself has recognized this fact, EPA's proposal falls short of what the scientific evidence requires. The Clean Air Scientific Advisory Committee recommended the EPA revise both the annual standard and the daily standard, but the EPA disregarded this advice, forcing the Scientific Advisory Committee to reconvene and reiterate its advice to the EPA.

The committee has made it clear that the EPA's proposal for the coarse particle standard was outside the bounds of scientific evi-

dence. If the EPA were to listen to the advice of its science advisors, tens of thousands of lives could be saved. The standard proposed by the EPA would result in a 22 percent reduction in deaths. Standards set within the range recommended by the Scientific Advisory Committee would save up to 48 percent more lives.

Particulate matter kills more people than HIV/AIDS and more people than drunk driving. It is a big killer and we need to reduce it as soon as possible. That is what the science shows.

Thank you. I look forward to hearing from the witnesses.

[The prepared statement of Senator Jeffords follows:]

STATEMENT OF HON. JAMES M. JEFFORDS, U.S. SENATOR FROM THE STATE OF VERMONT

Mr. Chairman, thank you for having this hearing on the EPA's proposed standards for Particulate Matter.

There is perhaps no more important environmental standard than the National Ambient Air Quality Standard for Particulate Matter. Tens of thousands of Americans die prematurely each year from particulate matter.

The NAAAQS are the cornerstone of the entire Clean Air Act. Their fundamental purpose is to tell us when the air is safe to breathe. For more than 30 years, these standards have been set based solely on health considerations, using the latest scientific evidence. Congress specifically chose to not allow consideration of costs when setting these standards.

Those who call for a cost-benefit analysis would have us set a standard that fails to protect the health of all Americans. We cannot afford the human cost of such an approach.

Consider this: If cost benefit analysis is used to set the NAAAQS and the compliance costs of a standard are estimated at $1 billion, the EPA would be required to set a standard that would allow up to $1 billion worth of people to die from air pollution. So just how do we measure $1 billion in human life? Setting the NAAAQS in that way is unconscionable. Telling people their air is safe to breathe, when it is actually not safe, is unacceptable public policy.

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The Committee also made it clear that the EPA's proposal for the coarse particle standard was outside the bounds of the scientific evidence. If the EPA were to listen to the advice of its science advisors, tens of thousands of lives could be saved.

The standard proposed by EPA would result in a 22 percent reduction in deaths, but a standard set within the range recommended by the scientific advisory committee could save up to 48 percent more lives. Particulate matter kills more people than HIV/AIDS and more people than drunk driving. It is a big killer and we need to reduce it as soon as possible. That is what the science shows.

Thank you, and I look forward to hearing from our witnesses.

Senator INHOFE. Thank you, Senator Jeffords.

Senator VOINOVICH.

OPENING STATEMENT OF HON. GEORGE V. VOINOVICH, U.S. SENATOR FROM THE STATE OF OHIO

Senator VOINOVICH. Thank you, Mr. Chairman. I appreciate the fact that you bumped this hearing from my subcommittee to the full committee. It shows how important this matter is. To sum up last week's hearing, we do not know the impact of revising the particulate matter standards, and let's review what we do know.

We know that the air is significantly cleaner; that the EPA has proposed to move the goal posts in the States as they implement the current standards. We believe that the Agency is under-

estimating the number of non-attainment counties. We know that the current non-attainment designations threaten highway funding and jobs and increase energy prices and that revised standards would exacerbate the situation.

We also know this decision has a profound impact on the health and well being of many, as the National Black Chamber of Commerce President Harry Alford stated, "The biggest health risk to African Americans anywhere is poverty." I will never forget when Tom Mullen from the Catholic Charities came here several years ago and testified that in setting ambient air standards and environmental policy, that we should take into consideration its impact on the poor and the elderly, and the Clean Air Trust named him villain of the month because he had the audacity to raise the issue.

Today's hearing focuses on the science and risk assessment behind the standards. I make three points. First, according to the recent proposal and risk assessment, we will hear today the EPA's estimates, the risk from exposure to fine particulate matter has declined from the level of risk estimated in setting the last standards.

Second, the health benefits of EPA's proposal are not fully understood. A 2002 National Academy of Sciences report made 34 recommendations to improve our understanding of the estimation of the public health benefits. According to GAO that we asked for, EPA has fully implemented less than 25 percent of recommendations made back in 2002 by the National Academy of Sciences. For example, the NAS, along with the Office of Management and Budget and EPA's Inspector General, has urged the Agency not to assume that all fine particulate constituents have the same potency. As stated in the IG's report, otherwise some facilities may install unneeded controls, while some needed controls may go uninstalled. Ultimately, compliance may be further delayed and more costly.

Unfortunately, the EPA has largely not implemented the recommendations. The standards should not be revised until we have adequate information that tells us with greater certainty the health benefits and whether we are targeting the most harmful constituents of particulate matter, especially considering the negative impacts on this country's economy. Air quality will continue to improve through implementation of the current standards and the Federal clean air rules.

I know you want me to cut this short. I will just basically say, Mr. Chairman, it is important that everyone understand that science can only take us so far, but ultimately it is a policy decision whether or not to change a standard. That is why the Clean Air Act states the air quality standards are to be set in the judgment of the Administrator. Given this judgment discretion, how can EPA revise the particulate matter standards when the public health benefit is not fully understood?

Thank you, Mr. Chairman.

[The prepared statement of Senator Voinovich follows:]

STATEMENT OF HON. GEORGE V. VOINOVICH, U.S. SENATOR FROM THE STATE OF OHIO

Mr. Chairman, after our hearing in the subcommittee, I appreciate this important issue being elevated to the full committee.

To sum up last week's hearing, we do not know the impact of revising the particulate matter standards. Let's review what we do know:

• We know that the air is significantly cleaner and that EPA has proposed to move the goal posts on States as they implement the current standards.

We believe that the Agency is underestimating the number of nonattainment counties and that Federal clean air rules will only mitigate this problem.

• We know that the current nonattainment designations threaten highway funding and jobs and increase energy prices and that revised standards would exacerbate the situation.

• We also know that this decision has a profound impact on the health and well-being of many, as National Black Chamber of Commerce President Harry Alford stated: "the biggest health risk to African Americans anywhere is poverty."

Today's hearing focuses on the science and risk assessment behind the proposed standards. I will make three points.

First, according to the recent proposal and risk assessment, we will hear today that EPA's estimate of the risk from exposure to fine particulate matter has declined from the level of risk estimated in setting the 1997 standards.

Second, the health benefits of EPA's proposal are not fully understood. A 2002 National Academy of Sciences (NAS) report made 34 recommendations to improve our understanding of the estimation of the public health benefits. According to the Government Accountability Office report that Chairman Inhofe and I requested, EPA has fully implemented less than 25 percent of them.

For example, the NAS along with the Office of Management and Budget and EPA's Inspector General has urged the Agency not to assume that all fine particle constituents have the same potency. As stated by the IG: "Otherwise, some facilities may install unneeded controls, while some needed controls may go uninstalled; ultimately, compliance may be further delayed and more costly."

Unfortunately, EPA has largely not implemented the recommendations. The standards should not be revised until we have adequate information that tells us with greater certainty the health benefits and whether we are targeting the most harmful constituents of particulate matter—especially considering the negative impacts on our economy. Air quality will continue to improve through implementation of the current standards and the Federal clean air rules.

EPA claims that they will address more of the NAS recommendations when the final rule is issued. At this time, we are told that there will also be a more complete Regulatory Impact Analysis and new science will be considered. It is unacceptable for the public and this committee to get critical information only after the final decision has already been made.

Third, testimony that we will hear today indicates that EPA selectively used study results in developing the proposal.

In conclusion, it is important that everyone understand that science can only take us so far, but ultimately, it is a policy decision whether or not to change a standard. That is why the Clean Air Act states that air quality standards are to be set "in the judgment of the Administrator." Given this judgment discretion, how can EPA revise the particulate matter standards when the public health benefit is not fully understood?

Mr. Chairman, I again thank you for holding this hearing and look forward to hearing from the witnesses. Thank you.—

Senator INHOFE. Thank you, Senator Voinovich.

Senator BOXER.

OPENING STATEMENT OF HON. BARBARA BOXER, U.S. SENATOR FROM THE STATE OF CALIFORNIA

Senator BOXER. Thank you, Mr. Chairman, for holding this hearing. I ask that my full statement be placed in the record.

Senator INHOFE. Without objection, so ordered.

Senator BOXER. I will read part of it.

This hearing is important because protecting the air that Americans breathe is important. It is a critical responsibility of this committee. We have made great strides in this country, but unfortunately EPA's proposed standards for controlling toxic soot and dust drastically depart from this path.

Now, I won't get into politicizing science, but what I want to say here is EPA has rejected advice from its own Clean Air Science Advisory Committee on health-based standards for toxic soot and

dust. This, despite the fact that the American Lung Association and other public health groups say the children, the elderly and people with heart disease, diabetes or respiratory diseases are especially vulnerable to the adverse impacts of such pollution.

Let me be more specific. Particulate pollution causes premature death, whether you are in Oklahoma, New York, New Jersey, Vermont, California or Ohio. Particulate pollution causes premature death. It exacerbates asthma, cardiovascular disease, including heart attacks and strokes, and it increases hospital admissions.

I have to say, sometimes in this committee we talk about things as if they were very interesting scientific issues. But the bottom line is what we are talking about here is the longevity or lack of such of the American people and the quality of their lives, be they children or adults. More than 160 national, State, local, environmental, religious groups, and I want to point out religious groups are getting involved in this, and public interest groups have urged EPA to increase these clean air protections. EPA's Children's Health Advisory Committee also urged EPA to revise these standards to protect children.

I would ask unanimous consent that these letters be placed in the record at this time. Mr. Chairman, I would like to place these letters from the religious groups and others into the record at this time.

Senator INHOFE. Without objection.

[The referenced letters can be found on pages 193-201.]

Senator BOXER. EPA has also proposed a protective standard for coarse particulate matter. The Agency proposal ignores pollution by exempting some industries, exempting them, industries such as mining. In addition, EPA proposes such a limited amount of monitoring that pollution will go undetected in many of our medium size and small cities.

So EPA must stop this politicizing. They are politicizing scientific decisions, and I resent it as a U.S. Senator from the largest State in the union, where we are deeply affected by this. A particularly egregious example was identified by Mr. Bart Ostro, chief of California's Office of Environmental Health, regarding the particulate pollution rule. On February 3, 2006, Mr. Ostro testified before EPA's Science Committee that "last minute additions of edits and opinions by OMB and others circumvented the entire peer review process." That is a condemning statement.

The Clean Air Act has it right. Sound science and the protection of public health should guide the establishment of Clean Air Act standards and sadly, Mr. Chairman, I am very worried about the fact that we seem to be veering away from science and making politics the key ingredient in these decisions.

Thank you.

Senator INHOFE. Thank you, Senator Boxer.

Senator DEMINT.

OPENING STATEMENT OF HON. JIM DEMINT, U.S. SENATOR FROM THE STATE OF SOUTH CAROLINA

Senator DEMINT. Thank you, Mr. Chairman. I am very interested in the results today. I appreciate our witnesses.

Last week, we looked at the possibility of a rule change. As we all know, the Clean Air Science Advisory Committee has recommended that EPA lower its annual standard for particulate matter. What I find interesting is that the air today is significantly cleaner than it was years ago, and even more intriguing is the risk assessment of health complications due to long-term exposure, $PM_{2.5}$ ambient air, have been reduced.

Studies have shown that the risk assessment for $PM_{2.5}$ have either stayed the same or been reduced. Then why is the EPA receiving a recommendation from the committee to tighten the standard? Obviously, I am very interested in clean air. I just want to make sure that we find the source and really do something that cleans the air, not just do something to do something.

The mechanisms that were used to direct the committee to recommend more stringent annual standards are actually disturbing. The EPA staff memo directed the committee to study three specific scientific studies. One of the studies gave mixed results of the human risk of high levels of $PM_{2.5}$; another was nullified by the scientists who performed the study because the data constantly changed based on their assumptions; and the third one was based on cities outside the United States over a 6-year process that showed a drop in ambient air quality.

So I am very interested in the testimony today. We want to know about good science. We want to know how to clean the air. I am just afraid we are looking at doing something just to look like we are doing something. We will end up costing this country a lot of jobs and prosperity, and not leave the air any cleaner.

So I am here to learn, and I thank you, Mr. Chairman.

Senator INHOFE. Thank you, Senator DeMint.

Senator LAUTENBERG.

OPENING STATEMENT OF HON. FRANK R. LAUTENBERG, U.S. SENATOR FROM THE STATE OF NEW JERSEY

Senator LAUTENBERG. Thanks, Mr. Chairman, for giving us another opportunity to examine the need to protect our air.

This week gives us an urgent reminder about what we have to do. The red air days are oppressive. I felt it myself, not enough to make me ill, but enough to make me realize that that air wasn't particularly clean.

Parents are warned not to let children play outdoors if they have respiratory problems because of increased danger of asthma attack or other complications. We know that particulate matter pollution triggers asthma attacks and affects the health of people with other respiratory and lung conditions.

Today, we are going to hear how it also poses threats to people with heart conditions. But we will hear a lot of talk about uncertainty. We are not sure. It is like saying, well, the fire is only in the basement; let's not get excited upstairs.

I heard the same thing 20 years ago when I wrote the law to ban smoking on airplanes. The tobacco industry said there was too much uncertainty about the effects of secondhand smoke. There is no doubt anymore about it. Today, we know that there is real danger from secondhand smoke and we are so grateful that we did something when we did it.

We have heard the same excuse about uncertainty for years in the discussion of global warming. Look outside; feel it. The claim of uncertainty is the smokescreen that diverts attention from real problems. It is merely an excuse to avoid taking any action that might cost an extra few bucks to accommodate our need.

We are here to represent the 25 million American adults with heart disease and 6 million children with asthma, one of whom is my grandson, Alexander. There is no uncertainty about whether they deserve clean, healthy air. This week, we learned of a report on particulate matter which had been requested by OMB. That report suggests that particulate matter is actually a worse problem than we previously thought. So much for uncertainty.

So we ought not to let a handful of particular interests pressure us into doing anything that will permit more harm to millions of Americans. I think that this is a matter of urgency and I think we have to get on to making sure that the standards that we have are the highest and are the toughest than we get to meet.

And not without consideration for either jobs or investment. But I will tell you, when we talk about jobs and I read the reports about what is happening, where things are being made today, they are not being made in India and other places, or Bangladesh because of environmental issues. They are being taken away because of cost issues. So we shouldn't confuse the facts.

I thank you, Mr. Chairman, once again, for the opportunity to air this problem.

Senator INHOFE. OK. Senator Clinton.

OPENING STATEMENT OF HON. HILLARY RODHAM CLINTON, U.S. SENATOR FROM THE STATE OF NEW YORK

Senator CLINTON. Thank you, Mr. Chairman.

Although we are making opening remarks, I want to begin by welcoming someone whom I have a great deal of regard for, Dr. George Thurston, to the committee. Dr. Thurston is on the faculty at New York University School of Medicine, where he conducts investigations into the human health effects of air pollution. He is truly one of the leading scientists in this field. I am pleased that he will be testifying on the second panel.

I have particular regard for Dr. Thurston because when I was raising questions about the quality of air after 9/11 and the Administration was assuring everyone that there were no problems in the air, and in fact everything was fine, Dr. Thurston was one of those who said this is just not squaring with the scientific work I have done.

Well, now nearly 5 years later, we have hundreds and hundreds of firefighters, police officers, construction workers, residents and others who are suffering from respiratory distress. Now, that was an intense experience because the air was so contaminated with everything that came from the World Trade Center. We can look at that and we can draw some conclusions about the impact of such a horrible event.

But it is also true that after 9/11 when all the planes were grounded, there are a lot of pictures showing how clean the air was. We are facing a very serious question here. This is an important issue, not just in New York, but across our country. Since the

1997 revisions to the particulate matter standards, numerous scientific studies have been published. These have not been partisan studies. These have not been ideological studies. They have ranged from scholarly academic studies like Dr. Thurston, to the American Lung Association.

These studies have overwhelmingly strengthened the links between particulate pollution and a range of adverse health outcomes, including asthma, heart attacks, hospital admissions, adverse birth outcomes, and premature death. Some recent studies have even linked long-term particulate matter exposure to increased risk of lung cancer.

In addition, our understanding of the biological mechanisms at work helps explain these links better than we understood back in 1997. So I was dismayed when the EPA disregarded the advice of its Clean Air Science Advisory Committee.

You know, when CASAC recommended what it thought needed to be done, the EPA decided not to lower the annual PM standard in its proposed revisions. This action prompted a letter back to the EPA from CASAC, which I think is unprecedented. That letter reiterated the CASAC's finding. All of these scientists who have no particular ax to grind, "the epidemiological evidence supported by emerging mechanistic understanding indicates adverse $PM_{2.5}$ health effects below 15 micrograms per cubic meter."

Now, I think the science is clear. One of the reasons the air is cleaner today is that for more than 30 years we have been cleaning it up. So we have a little bit in the bank, but that is not going to stand.

So Mr. Chairman, I hope that we will go with the lower standard, and I ask unanimous consent to submit the full statement to the record.

Senator INHOFE. Without objection, so ordered.

Senator INHOFE. Senator Carper.

OPENING STATEMENT OF HON. THOMAS R. CARPER, U.S. SENATOR FROM THE STATE OF DELAWARE

Senator CARPER. Thanks, Mr. Chairman.

I want to follow up on what Senator Clinton has said, if I may, and also just kind of respond to what Senator DeMint has said. EPA doesn't come up with these recommendations out of thin air. They have this CASAC committee that Senator Clinton just referred to. They made their recommendations for advising the annual and the daily limits on this particulate matter.

Good people, they are all scientists. They have no ax to grind. What they try to do is to use good science and to give us their best recommendations, and also to give those recommendations to EPA. EPA has reviewed, I am told, over 2,000 scientific studies I believe since 1997, and they found an association between particulates in ambient air and all kinds of health problems. We have heard a couple of them, a recitation of them from Senator Clinton. I want to mention them again: aggravated asthma, chronic bronchitis, reduced lung function, irregular heartbeat, heart attacks, and premature death in people with heart or lung diseases.

In just nine cities that EPA looked at, particulates would cause an estimated almost 5,000 premature deaths unless current stand-

ards are strengthened. EPA could have come in and frankly followed the advice of CASAC. They didn't. They erred the other way. For some of us on this committee, we were disappointed with that result.

I do want to commend Administrator Johnson for acknowledging that the science does support strengthening the current particulate matter standards. I am concerned that the proposed standard is higher than the range recommended by EPA's CASAC. I look forward to hearing today EPA's justification for that decision.

Finally, I would like to follow on a point made during last week's hearing by a number of Senators, as well as Bill Wehrum of EPA. To do so, let me just quote Mr. Wehrum's testimony from last week. Here is what he said: "between 1970 and 2005, gross domestic product increased by 195 percent. Vehicle miles traveled increased 178 percent. Energy consumption increased 48 percent. The U.S. population grew by 42 percent. During the same time period, total emissions of the six principle air pollutants dropped by 53 percent."

In other words, since the inception of the Clean Air Act, our country has made significant increases in our energy usage, mobility and an ever-growing economy, and at the same time we have seen our air pollution decrease. Our Nation's air has not become cleaner on its own. The improvements are a direct result of specific air regulations and the implementation of the National Ambient Air Quality Standards. The arguments we are hearing today that claim that tighter air regs will hurt our economy and cost people jobs are not new. We have heard these claims every time EPA has proposed a new regulation over the past 30 years.

However, as Mr. Wehrum and others on the committee correctly pointed out last week, those claims have not proven to be true. We can have cleaner air. We can grow our economy and we can do those things today, as we have in the last 30 years.

Thank you, Mr. Chairman.

Senator INHOFE. Thank you, Senator Carper.

That will conclude our opening statements. We have on our first panel Dr. George Gray, Assistant Administrator for Research and Development for the EPA; and John Stephenson, Director, Natural Resources and Environment for the United States GAO.

We will start with you, and try to confine your remarks to 5 minutes, if you could. Dr. Gray, you are recognized.

STATEMENT OF GEORGE GRAY, ASSISTANT ADMINISTRATOR FOR RESEARCH AND DEVELOPMENT, U.S. ENVIRONMENTAL PROTECTION AGENCY

Mr. GRAY. Thank you, Mr. Chairman, members of the committee. I am George Gray, the Assistant Administrator for the Office of Research and Development at the Environmental Protection Agency.

I want to thank you for the opportunity to discuss EPA's evaluation of the scientific evidence for potential health effects of airborne particulate matter known as PM. Last week, the subcommittee heard from my colleague Bill Wehrum, the Acting Assistant Administrator for Air and Radiation and the EPA's review of the National Ambient Air Quality Standards, we call them the NAAQS.

During the testimony, he explained to you the crucial role that science plays in helping to inform our decisions about the NAAQS. Today, what I would like to talk to you about in more detail is the science, how it is prioritized, how it is developed, and how it is synthesized and integrated.

As the Assistant Administrator for the Office of Research and Development, I am responsible for both the development of new scientific information targeted at specific Agency needs, and for the evaluation, synthesis and integration of the world's peer reviewed literature and to a document that informs EPA decisionmakers.

As we characterize the current state of our collective scientific knowledge, we are careful to point out the strengths and weaknesses of this large body of information so that informed decisions can be made. It is clear that scientists and staff of EPA play a crucial role in the development and evaluation of the world's scientific information to inform their review of the NAAQS.

A very important function of EPA is the synthesis and integration of these thousands of individual acts of science to provide a clear characterization of our knowledge and the degree to which we are still uncertain about some aspects of PM health and environmental effects.

We have a scientifically rigorous process by which we evaluate and interpret this important body of knowledge and we ensure that our interpretation of them is complete, transparent, unbiased, and consistent with an array of views in the scientific community.

A fundamental step in the review of the NAAQS is the evaluation of scientific evidence in the preparation of scientific assessments known as criteria documents by the National Center for Environmental Assessment, which is part of the Office of Research and Development. The development of criteria documents involves a review of thousands of peer-reviewed research publications, evaluation of those studies that are most relevant to the review of air quality standards and the integration of scientific information across disciplines.

The body of evidence must be reviewed, evaluated, weighed and then accurately and objectively described to help inform our decisions about National Ambient Air Quality Standards. For the current PM review, EPA evaluated research studies that addressed a wide range of issues, including PM toxicology, epidemiology, atmospheric chemistry, human exposure and other areas of environmental effects. Thousands of studies were reviewed and over 2,000 were referenced in the final criteria document, many of which were conducted or funded by EPA's Office of Research and Development.

Considered together, these new studies significantly advanced our understanding of PM's potential effects on public health and welfare and reduced the uncertainties associated with some important aspects of the science. Drawing on the evaluation of studies reviewed in the PM criteria document about health effects and dose response, as well as information about exposures to PM, EPA also completed a risk assessment to estimate the degree to which various approaches for revising the standards would potentially affect the public health risks that are posed by PM.

Further, the Agency prepared a document known as a staff paper that utilized the evaluation and characterization of scientific evi-

dence in the criteria document, together with the results of the risk assessment, to help inform policy judgments that are required in making decisions about the NAAQS.

In summary, the Bush administration is committed to the development and use of the highest quality scientific information to inform decisionmaking. The mission of the Office of Research and Development is to develop, evaluate and communicate relevant scientific information to the Administrator and to assure that the Administrator is well informed of the nature, the strengths, and the limitations of this information. EPA has sponsored a targeted and effective research program on particulate matter, and I am pleased to convey to you and others the value of this investment.

We have made a great effort to evaluate and characterize the existing and new scientific results available on particulate matter and I am personally pleased to share with you my views on this work. I look forward to addressing any questions that you may have, and I respectfully request that my full written testimony, as submitted, be inserted into the record.

Senator VOINOVICH. [Presiding] Thank you, Mr. Gray.

Mr. Stephenson, before you give your testimony to the committee, I am going to ask that this report by the GAO be made a part of the record. I look forward to your testimony.

[The referenced document can be found on pages 86-90.]

STATEMENT OF JOHN STEPHENSON, DIRECTOR, NATURAL RESOURCES AND ENVIRONMENT, U.S. GENERAL ACCOUNTABILITY OFFICE

Mr. STEPHENSON. Thank you, Mr. Chairman and other members of the committee. I am pleased to be here today as the committee considers the science and risk assessment supporting EPA's proposed revisions to the national air quality standards for particulate matter.

A large body of scientific evidence over the past several years links exposure to particulate matter to serious health problems, including asthma, chronic bronchitis, heart attack and premature death. EPA, as part of its authority under the Clean Air Act to periodically review and revise as appropriate the air quality level at which to set national standards, proposed revisions to the particulate standards. It issued a draft regulatory impact analysis in January, 2006 of the revisions' expected costs and benefits.

As you know, EPA's estimates of the expected benefits from its air pollution regulations have in the past often been controversial and the methods the Agency used to prepare these estimates have been questioned. As a result, at the direction of the Senate Appropriations Committee, the National Academy of Sciences evaluated EPA's overall methodology and approach for estimating the health benefits of all proposed air regulations, and in 2002 issued a report that made recommendations to EPA to, among other things, conduct more rigorous assessments of uncertainty, increase the transparency of how it estimates benefits, conduct more detailed analysis of exposure, and estimate the benefits of each regulatory option under consideration.

Mr. Chairman, you asked GAO to determine the extent to which EPA has implemented the academy's recommendations and our re-

port that you just mentioned being released today provides a detailed discussion of each recommendation and EPA's efforts to respond to them.

In summary, we found that while the National Academy generally supported EPA's overall approach to estimating benefits, it made 34 specific recommendations for improvements. EPA is making progress in responding to the National Academy's recommendations, but in the case of its January, 2006 analysis, EPA had applied 8 of the recommendations, partially applied 14, and did not apply 12.

For example, in response to the Academy's recommendations, EPA evaluated how benefits might change, given alternative assumptions, and discussed sources of uncertainty not included in the benefit estimates. Although EPA applied an alternative technique for evaluating one key uncertainty that concerning the causal link between exposure to particulate matter and premature death, the health benefits analysis did not assess how the benefit estimates would vary in light of other key uncertainties, as the Academy had recommended.

Consequently, EPA's response represents a partial application of some of the recommendations. EPA told us that insufficient resources impeded its progress in applying some of the Academy's recommendations, citing in particular the limited availability of skilled staff, time and other resources to conduct the recommended analyses. EPA also stated that in some cases, it did not believe the state of the scientific knowledge was sufficient to implement other academy recommendations. One such area has been mentioned today, is for EPA to determine the relative toxicity of particulate matter components. EPA intends to pursue research and development so that additional academy recommendations such as that can be incorporated in future air regulations.

We believe that continued commitment and dedication of resources will be needed if EPA is to fully implement the improvements recommended by the National Academy. In particular, EPA will need to ensure that it allocates resources to needed research on emerging issues such as the relative toxicity of particulate matter, and to assessing which sources of uncertainty have the greatest influence on the benefit estimates. EPA officials said that they expect the final rule, due September 27, will better address other academy recommendations pertaining to uncertainties associated with the health benefits estimates.

We should all be closely examining EPA's final rule because a more robust analysis of the remaining uncertainties, not addressing the proposal, will be critical if decisionmakers and the public are to better understand the likelihood of actually attaining the health benefit estimates.

Mr. Chairman, that concludes my summary. I will be happy to answer questions.

Senator VOINOVICH. [Presiding.] Thank you very much.

In our last hearing, we talked particularly about asthma. I would like to read a statement to both of you and have you comment on it in terms of what impact do you believe that increasing the particulate role would have on the issue of asthma.

According to the Centers for Disease Control and Prevention, from 1980 to 2001, the number of Americans with asthma tripled to 20.3 million. From 1975 to 2000, the number of asthma attacks also tripled. At the same time, EPA data indicates the emissions of the six principal air pollutants dropped by 53 percent since 1970. Thus, while the number of people with asthma and the incidence of attacks have increased, air pollution has decreased substantially over the same time.

Senator Lautenberg stated at the hearing, "But during the summertime when most people are enjoying their vacation playing outside, children with asthma often have to stay indoors, and the reason that they have to stay indoors is the air is just too dangerous for them to breathe."

However, staying indoors is exactly the opposite of what children should be doing, according to a report on the indoor air pollution. EPA "studies of human exposure to air pollutants indicate the indoor levels of many pollutants may be 25 times and occasionally more than 100 times higher than outdoor levels. These levels of indoor air pollutants are of particular concern because it is estimated that most people spend as much as 90 percent of their time indoors. The poor quality of indoor air and the amount of time spent indoors seems to explain the increased incidence of asthma better than outdoor air which is greatly improved."

Furthermore, according to NIH, air pollution is one of the many things that can bring on asthma symptoms. They talk about animal dander, dust mites, cockroaches, pollen and mold, cigarette smoke, air pollution, cold air, strong odor, scented products, strong emotional expression and stress, medicines, sulfites in food, or beverages.

Will you comment on how you think, if we improve this particulate matter, it is going to make a difference in terms of something like asthma, that is always the subject matter here before this committee?

Mr. GRAY. Senator, as many of you have stated, it is very clear that the issue of particulate matter and the ambient air quality standard is one of the most important decisions that EPA makes. Particulate matter is linked with a variety of adverse health outcomes, exacerbation of asthma being one of them. That is, no one is suggesting that in fact particulate matter causes asthma, but that it may be something that helps to trigger an attack or make them worse.

That is part of the evidence, along with the other health effects that we can consider. They were part of the rationale behind our proposed rule that you can read about, where we suggested a particulate matter ambient air standard. We also asked for people's views. We asked for input from the scientific community and from the public about that. The information we are going to get is going to be an important part of the ultimate decision we make. We are trying to understand the range of scientific information on all of the health effects of particulate matter, and the role that that might play in the ultimate judgment of an appropriate standard.

Senator VOINOVICH. Mr. Stephenson, the fact that your report says that the recommendations of the National Academy of Sciences in terms of the things that you need to do to measure

whether or not particulate matter has an impact on health have not been carried out by the Environmental Protection Agency. Do you believe that they have enough information now that they can intelligently, objectively make a decision in terms of what this rule's impact will have on public health?

Mr. STEPHENSON. Well, one of the problems is the rulemaking process doesn't lend itself to a broad picture of air quality in general. We are looking at particulate matter in this case. Our concern was that many of the uncertainty analyses were done, but they were never rolled up into a more robust analysis of all the uncertainties such that you could determine the ranges of exposure and project the health benefits in that way.

So we think they have data. It is a question of how you analyze that data.

Senator VOINOVICH. Do they have enough data to intelligently, objectively decide whether or not for public health, and I am not talking about weighing all the other sides of this. In other words, they are not supposed to weigh the impact on the economy and jobs and poor people's energy costs and all the rest of that. They close their eyes to that. But do they have enough information to intelligently, objectively make a decision in terms of what impact a new rulemaking it more stringent would have on public health?

Mr. STEPHENSON. In some cases yes and in some cases no. They need more exposure data. They need more data to determine how particulate matter affects various populations, how people inhale the particulate matter, etc. There is another category, which you mentioned, on the components of particulate matter, what makes up particulate matter. As Chairman Inhofe mentioned, asbestos is certainly more dangerous than dust. There is more research that is needed in that area in order to determine if there is in fact a difference between different types of particulate matter.

Senator VOINOVICH. Thank you.

At the request of the Ranking Member of the subcommittee, he asked that Senator Boxer be next.

Senator BOXER. Thank you very much. I appreciate that.

I really have one question that I want to ask to both of you. Assistant Administrator Gray, EPA's proposal to reduce dangerous levels of coarse particulate matter or toxic dust relies on air pollution monitoring to detect potential violations. However, the President's proposed budget for fiscal year 2007 cuts these monitoring funds by $17 million. State and local Clean Air Act administrators believe this will severely weaken monitoring programs and likely result in significant staff cuts across the country, so we will not have the people necessary to tell us what this monitoring is showing us. And what the monitoring is showing us is very important because at certain levels, we know our most vulnerable populations, our children, our elderly, our sick, our weakest, are impacted.

So I don't quite get why we see that kind of a cut, $17 million is not a lot in the scheme of things, when you think of what we are spending in Iraq every week. It is in the billions. We need the $17 million to be restored. It just boggles the mind that would be gone.

Let me tell you the impact of it that we are being told. We are being told that as a result of this, dangerous pollution will go undetected in rural areas and cities, such as California's San Joaquin Valley where we have our farms and workers, farm workers; Missoula, MT; Carson City, NV; the Raleigh-Durham metro area in North Carolina; the Fort Worth-Arlington area in Texas.

I am just giving you examples of cities and regions that don't meet the population set out here. So I am wondering if you think that people in these areas deserve less protection than people in larger areas. To Mr. Stephenson, given the same set of facts, you have testified before that EPA fully or partially implemented only two thirds of the National Academy of Sciences' recommendations, but that Agency officials complained of a lack of funds hampered their ability to do so. Your testimony states that continued commitment and dedication of resources will be needed if EPA is to fully implement the improvements recommended by the National Academy.

This is an Administration, and my colleagues on the other side and we join them in this. We want to do everything that is based on science, and the National Academy of Sciences puts forward the plans, and then we don't have the funding. So this $17 million cut that I am coming back to, and then there is $1 million in cuts to EPA's budget for science and research for developing and implementing clean air standards.

So I would like both of you to react to these budget cuts, and how do you, first of all defend them, if you do, Mr. Gray and Mr. Stephenson. Can you just give us some straight talk about what it really means to our people?

Mr. STEPHENSON. Well, let me just say that we have in the past recommended in other reports on other air issues that more monitoring is needed. We are a data-based organization. We support the need for real monitoring data. That is the cornerstone of scientific research. So if there is a cutback in the amount of data you actually collect, we would see that as a problem.

Senator BOXER. And Mr. Gray, I assume you don't think it is a problem?

Mr. GRAY. I want you to know that the EPA is committed to having the information that they need to make good science-based decisions. In fact, it is our network of thousands of monitors that we operate, together with our partners in the States, that helps give the public information Senator Lautenberg referred to, when there is a Code Red day. That information comes from monitors that are like those or even perhaps from those that are maintained by the Agency. But it also helps to support the research that we are talking about.

Senator BOXER. Wait a minute. Excuse me for interrupting you.

Mr. GRAY. Yes.

Senator BOXER. We agree that monitoring is important. I am asking you to tell me why, then, would you be cutting that budget?

Mr. GRAY. First of all, we have invested over $100 million since 1998 in monitoring and specifically in monitoring speciation, questions that we are being asked to address by the National Academy, by this committee and by others. In addition, we believe that we can find efficiencies. We can do our monitoring in better ways. We

are all being asked to do things more efficiently and we are looking for efficiencies.

Senator BOXER. So you don't believe that the local people who are telling us that as a result of these cuts there will be pollution undetected in rural areas and cities, including for example, San Joaquin Valley in California; Missoula, MT; Carson City, NV; Raleigh-Durham metro. We have a whole list of them. You think that is an overreaction and that these people will in fact have their air monitored? Is that what you are telling us?

Mr. GRAY. I am telling you that I am sure that they may be concerned, but that we are committed to making sure that we have the information that we need to monitor the air, to make sure that everyone has an adequate level of protection.

Senator BOXER. So they may be concerned, but they shouldn't be concerned because you are going to monitor their air. Is that what you are saying?

Mr. GRAY. I am saying that.

Senator BOXER. Yes or no? Are you going to monitor the air in those areas and those regions?

Mr. GRAY. Most of the monitoring that is done is not done by the Agency.

Senator BOXER. Yes or no? Are you going to monitor the air in those regions?

Mr. GRAY. We are going to do what we need to do to make sure that we have the information.

Senator BOXER. Mr. Chairman, they are not going to monitor the air in these areas. You can't get a yes or no answer. It is very disturbing. Yes or no, can't get it.

Thank you.

Senator VOINOVICH. Thank you, Senator Boxer.

For the record, I would like insert a letter signed by 33 members of the U.S. Senate to the Appropriations Committee asking that they restore this $33 million to your budget, $17 million of which is to be used for monitoring. I think it is outrageous that some of the domestic budgets of this Government have not been given the dollars they need to get the job done that they are being asked to do. The non-defense discretionary budget is being clobbered, and we just stick our heads in the sand and ignore it.

If we ask you to do a job, you ought to have the money to get the job done and we are not giving you the money and the Administration should wake up to the fact that we have a problem not only in the EPA, but across the board. I am sorry.

Senator ISAKSON.

Senator ISAKSON. Thank you, Mr. Chairman.

On that subject, just to help educate me, this is a prospective cut that is being discussed based on last year's funding and this year's appropriation. Do you feel like you have sufficient funding at this year's level to do the monitoring necessary to make a reasonably informed determination on your standards?

Mr. GRAY. The information that we use to make judgments about our standards is based on a range of scientific information. It includes not only this monitoring.

Senator ISAKSON. No, no, no. Excuse me for interrupting. But in terms of funding for monitors, which was the question, I am just

wondering if you think you have enough funding at current levels, not contemplating the future proposed cut, to make reasonable determinations?

Mr. GRAY. We believe that we have the information that we need to make the determinations that we need to make.

Senator ISAKSON. OK. Second, the GAO report, and excuse me, but I will read this so I will get it correct, in referring to the unexecuted 12 recommendations where you all cited insufficient resources, the GAO report says, the Agency will need to ensure that it allocates resources to needed research on emerging issues such as the relative toxicity of particulate matter components and to assessing which sources of uncertainty have the greatest influence on benefits.

This portends that you have some discretion in allocation that you could use to move moneys to complete all the 34 recommendations that GAO recommended. Is that correct?

Mr. GRAY. We take the recommendations from the National Academy of Sciences very seriously. Characterizing the science well in a balanced way and characterizing uncertainty are very important to the Agency. I, in fact, take some comfort in the fact that we have achieved, at least partially two thirds of those, and when our final RIA comes out in September, I think you will find we have made even more progress.

Some of the recommendations will require additional work. They will require us to do methods development. They will require us to do research. At this point, for example, we have committed over $150 million for research to help address the question of speciation, one of the very highest priorities that we have in addressing National Ambient Air Quality Standards. So we are setting our priorities to help to meet many of those recommendations that came from the National Academy.

Senator ISAKSON. Should you implement a rule before you have reached all the conclusions you need to reach based on the studies of the science?

Mr. GRAY. I think that the recommendations from the National Academy are important for us in the way we do our Regulatory Impact Analyses. Those are very important documents that help the Nation, that help you understand the costs and benefits of the actions that we take. We cannot consider those in setting our National Ambient Air Quality Standard.

Senator ISAKSON. You cannot consider them?

Mr. GRAY. We cannot consider the costs in setting our National Ambient Air Quality Standard. For that reason, we evaluate the scientific evidence. We consult with the scientific community. We have our panel of experts that we use, and that is the information that goes into the setting of the National Ambient Air Quality Standard.

Senator ISAKSON. One final question, and I want to thank the Chairman for holding this hearing and the hearing we held I guess last week, wasn't it?

At the hearing last week, we had a county commissioner from Georgia, Walker County, GA, that is in non-attainment; entered into a compact with EPA; has from what I can understand from EPA and them, done everything that was asked of them within

their control. But they find themselves the recipient of pollution from other continents, as well as larger cities near them, and also Mother Nature with the Bermuda High which over the southeast traps so much of this stuff for about 5 months out of the year.

I know everybody has heard this before, but just one more time. It seems to me like the restrictions and the punishment, if you will, that somebody who has done everything admittedly within their control to do, that we are punishing the wrong person. There ought to be some attention to the unintended consequences of them receiving this pollution without any ability to stop or divert it after they have taken care of all of the things at the local level they need to do.

So I know there was some testimony by one of the gentlemen testifying that there is a way out through appeal to EPA? I want to just perfect the record from last week. Walker County, at their own expense, did extensive studies to do that, and demonstrate the point source of this pollution, but still was unable to get any breathing room from the EPA restrictions.

So I think we do, I am very much for clean and cleaner air, and I am very much for doing everything we can in terms of standards to implement that. But I also am for reasonable common sense on the application, particularly of these non-attainment standards when there is no control by the people who are being restricted, and in my case it is 63 percent of my State.

Thank you, Mr. Chairman.

Senator VOINOVICH. Thank you, Senator.

Senator LAUTENBERG.

Senator LAUTENBERG. Yes, thanks, Mr. Chairman.

Mr. Gray, the EPA is proposing to attain an annual health standard of particulates that is much less protective than that which California has adopted. Can you explain why?

Mr. GRAY. In the case of our proposed rule, first, it is important to note that we are committed to making sure that our National Ambient Air Quality Standards fulfill the Clean Air Act goal of protecting human health. Setting a standard, as we have already discussed, is a judgment of the Administrator. The job of my office, and a job that I think we have done very well, is to present the Administrator with a picture, a complete picture of the science, its strengths and its limitations, and the uncertainties of what we know and don't know, and to help inform that judgment.

In our proposed rule, we did describe the basis for the level of 15 that we proposed, but we did ask for comments on other levels. I want you to know that those comments are going to be taken seriously and they will be an important factor in the Administrator's decision. We are in the middle of an open-ended, deliberative process that is going to result in a final standard in September. It will fulfill that goal of protecting human health, taking into account the science that is available, the advice of our advisory committees like CASAC, and public comment.

Senator LAUTENBERG. Do you think that we are at a point in time where we think that there ought to be any making the standards more rigid, to raise the standards for particulate matter that we ought to move on with? Have we done enough research, in your judgment, Mr. Gray, to say that there are significant problems with

the ambient air quality as we know it? We ought to, particularly as it affects particulate matter, should we strengthen those standards, make them more rigid than they are? Or should we reduce them?

Mr. GRAY. Again, this is an important judgment that has to be made. National Ambient Air Quality Standards for particulate matter are some of the most important decisions that are made by the Environmental Protection Agency and the decisions are not made lightly. They are made with as much scientific information, as much scientific advice, as much scientific input as we can muster. That information is put before the Administrator in a balanced way, in a careful way, describing the strengths and limitations to help inform the judgment about whether the standard needs to be retained, needs to be raised, or needs to be lowered.

Senator LAUTENBERG. Do you make recommendations when you submit it to the Administrator as to the direction that EPA ought to take? Or is this simply, I've done my job and here it is? Or do you do all the decisionmaking as to the Administrator?

Mr. GRAY. My office does not do that. We prepare what we call the criteria document, thousands of pages of scientific information summarizing what we know and don't know. There are recommendations that come from the staff in the Office of Air and Radiation in EPA that make suggestions to the Administrator. CASAC makes suggestions to the Administrator. All of that informs their judgment.

Senator LAUTENBERG. You are aware of those recommendations?

Mr. GRAY. I am very aware of them, yes.

Senator LAUTENBERG. What is the conclusion that you would come to, hearing their recommendations? Do you think that the standards ought to be reduced, lowered?

Mr. GRAY. I think that when we put out our proposed rule and our proposed standards, we very clearly articulated the thinking behind them, the science behind them. We asked for public comment, scientific information that might be out there that we might find useful. All of that will inform our judgments.

Senator LAUTENBERG. I understand the mechanics, but there must be some conclusions out of this that say we could do better. We certainly ought not to do worse. The Chairman talked about a statement I made about keeping kids indoors. So I think I can come up with a conclusion that you have to choose your poison. In other words, if a mother unwittingly says, well, I am going to bring my child inside, or keep my child indoors because I think that the air quality is better. Here we come up with a conclusion that no, that there is more danger indoors than there is outside.

So that is quite a dilemma. I think that what we ought to do is understand people's emotion, and understand what people's thinking is. In the house, air conditioned, etc., and you think that the air quality is better. I know it's easier to breathe when you are in a house that has some air quality action going on.

So maybe we ought to meet outside in the interest of safety for the Senators and the visitors.

[Laughter.]

Senator VOINOVICH. Thank you, Senator.

Senator CHAFEE.

Senator CHAFEE. Thank you, Mr. Chairman.

Senator Isakson was talking about his county in Georgia, Walker County, and he mentioned particulate matter coming from other continents. In your studies, are there any other countries that are similar to us in our research? I don't want to put you on the spot, but that we can work with as we try and wrestle with reconciling the health benefits toward some of the regulations on particulate matter?

Mr. GRAY. There are two parts here. One is that we, as EPA, review the scientific information that is available to us to help us understand the appropriate levels that inform the judgments about the appropriate levels of air quality standards, we use information from around the globe. We use research that is done in the United States, Europe, and North America. We tend to focus on certain areas, partly for technical reasons. There is better monitoring data in the United States than in Canada, or monitoring is done differently than it is in other parts of the world and interpretation can become a little tricky.

We work with international experts. We work with international data to try to inform our decisions, to bring the best information to bear. In implementation, we do have to think about the fact that some of these pollutants can travel a very long way, when we think about how we control them.

Senator CHAFEE. Any countries in particular really leading the way, that we work more closely with? Or are we more on the forefront?

Mr. GRAY. Well, I don't know that I can speak to that very authoritatively, though I will say that in many cases we do think about international transport of pollutants, and we have efforts under way in EPA to help reduce the pollution that comes out of power plants in China because we know that that can affect air quality in the United States. So we are thinking about the global environment and trying to transfer some of the practices that we have to other countries not only to benefit them but to benefit us.

Senator CHAFEE. Again, I don't want to put you on the spot, but I am just curious. Are there any other countries that have stricter standards than we do?

Mr. GRAY. There are countries that have mixes of standards some parts of which are stricter than ours, and some they are not. In some parts of the world, even in some parts of this country, they may have standards that are very strict, but the rules for implementation are not as strict. When we set a rule, we mean it and it is going to mean something. That is not always the case; for example, the World Health Organization has set a standard that is stricter than ours, but it is completely aspirational and has no teeth to it.

Senator CHAFEE. Can we learn from some of the economic impacts from those stricter standards?

Mr. GRAY. Again, because many of them are not being implemented, because they are aspirational, it is hard to learn. If there are opportunities to learn, we will use them. As I said, we take our requirement for doing a Regulatory Impact Analysis very seriously. We want to make sure that we inform the Nation and you and any-

one else who is interested in it, of the costs and the benefits of these actions that we take.

Senator CHAFEE. OK, thank you.

On another subject, does the science dictate that the Administrator, I think this is following up on Senator Lautenberg's question, dictate that the Administrator choose a specific number in setting the particulate matter standards? Does the Administrator have some leeway?

Mr. GRAY. It is my understanding—and again I am a toxicologist, not a lawyer—that in the Clean Air Act recognizes that in fact, setting a National Ambient Air Quality Standard is a judgment on the part of the Administrator that considers the science, scientific advice, and a variety of other factors. So in fact setting a national ambient air quality standard is a judgment. In the Office of Research and Development, we do our very best to make sure that he has an accurate, unbiased characterization of the science to inform that judgment.

Senator CHAFEE. Thank you, gentlemen.

Thank you, Mr. Chairman.

Senator VOINOVICH. Senator Clinton.

Senator CLINTON. Thank you, Mr. Chairman.

Mr. Gray, in your testimony you said that EPA has spent $500 million since 1997 to improve our scientific understanding of the health impacts of particulate matter. Now, it is my understanding, based on all of this research that has been conducted since 1997 and the extensive review that the research has received from outside experts, as well as the public, that there is a consensus.

Let me ask you, does EPA have significantly more confidence now that current levels of PM are responsible for very serious health effects, including premature death?

Mr. GRAY. When we look at the science that is available to us, especially that which has been developed since the last evaluation of the standard in the late 1990's, I can say that there is better causal evidence linking particulate matter to a range of adverse health effects. The understanding of the levels at which that happens is much less clear.

Senator CLINTON. Right, but we actually know more today about the causal effects than we did in 1997. Is that correct?

Mr. GRAY. Based on what we have learned from epidemiology, from studies in people, from toxicology, it is believed we have a better understanding of the causal relationship, yes.

Senator CLINTON. What I am confused about is that given our understanding of the causal effects, which is greater than it was in 1997, given the recommendations from CASAC and others, in your proposed rule, EPA itself said that risk assessment was too uncertain for standard-setting purposes. What is the basis for that assertion?

I know that we are searching for the best way to implement a rule that does take into account the scientific evidence, particularly the causal connections, so how did EPA reach a different conclusion than all of the other experts who have reviewed this matter?

Mr. GRAY. I think an important point is that EPA doesn't come to a different conclusion on this issue of causality, is there a relationship between particulate matter and adverse health effects.

Where there is uncertainty and where the Agency felt there was too much uncertainty to rely upon the risk assessment for setting a standard is in the ultimate understanding of the strength of those relationships and the levels at which the effects occur.

Senator CLINTON. Well, then may I ask you, because we are limited, this is a very frustrating experience asking difficult question in 5 minutes, but may I ask you then, why wouldn't our Government err on the side of health? If there is an uncertainty of the mechanism, why wouldn't we go with the stricter standard as recommended by the weight of scientific evidence?

Mr. GRAY. I think I would echo your final sentence, that is the importance of the weight of scientific evidence. What we have done in our proposed rule is lay out what we think the scientific evidence is for the standards that we proposed. We asked for further comments, recognizing that others, reasonable minds can differ on this very large and very complex body of information. We have asked for comments. We have asked for further information, and that is all going to play a role in the final judgment of the Administrator for the final standard that will be coming out in September.

Senator CLINTON. Mr. Gray, may I also ask you, have you or anyone on your staff had contact with or from anyone in the White House concerning this proposed rule?

Mr. GRAY. In the preparation of the science here, the work that we do in the Office of Research and Development has been done in a completely public and open way through the CASAC process. All of our CASAC meetings are open to the public. All of their deliberations are public.

Senator CLINTON. I know, but have you or anyone on your staff had any contact with or from anyone in the White House with respect to this proposed rule?

Mr. GRAY. I can speak for myself and say that I have not. In terms of the science here, I cannot speak for my staff. I don't know.

Senator CLINTON. OK. But when you say in terms of the science that is a blanket denial of any contact whatsoever from or through the White House?

Mr. GRAY. It is important to recognize, and I have said this——

Senator CLINTON. Can you answer that yes or no?

Mr. GRAY. These are very important standards and that there are interagency reviews.

Senator CLINTON. Mr. Gray, I just want a yes or no answer.

Mr. GRAY. Our scientific information is in our hands.

Senator CLINTON. I know, but you haven't answered my question. Have you or anyone on your staff had any kind of contact with from or through the White House with respect to this proposed rule?

Mr. GRAY. In respect to the proposed rule, yes, of course we have.

Senator CLINTON. OK.

Mr. Chairman, could we get further information about this? I asked because, again, I go back to the very difficult experience I had after 9/11 when it became clear that the White House was directly interfering with the EPA scientific assessments and the public information provided to my constituents. I think that this committee deserves to know what, if any, kind of contact, pressure or

other relationship exists between the White House and the EPA with respect to this proposed rule.

Senator VOINOVICH. I would like to comment that I was just concerned about that as you are. I would really like to discuss that because I was not aware of the fact that the White House had had anything to do with whatever they came out with after that. I know one thing, that when Joe Albaugh came before this committee, no one seemed to know what was going on, and that was one of the things that disturbed me. That is why our legislation is so important. The President can move in and immediately determine what folks are exposed to.

I do not see that there is any problem with it, that we look into that. I would be surprised if there wasn't. I suspect once you are done with this, don't you have to submit it to OIRA to look at the cost-benefit analysis at OMB?

Mr. GRAY. As I said, with our proposed rule, there is clearly contact with the other parts of the executive branch.

Senator VOINOVICH. I think if you talk about the official contact, I think it is reasonable that we get an answer to that question.

Mr. GRAY. There is an interagency process that we use with all of our rulemakings.

Senator VOINOVICH. Why don't you share it with us, OK? Then I think we will feel a lot better about it.

Mr. GRAY. Certainly.

Senator VOINOVICH. Respond to Senator Clinton's concerns.

Senator CARPER.

Senator CARPER. Thanks, Mr. Chairman. Mr. Chairman, let me ask a first question of you. Do you recall several years ago when, maybe 3 or 4 years ago when Governor Whitman was EPA Administrator that you and I collaborated on a proposal calling for the establishment of a position within EPA?

Senator VOINOVICH. Here it is.

Senator CARPER. The Deputy Administrator for Science and Technology. There you go. You may have been talking about this when I was out of the room.

Senator VOINOVICH. No, we didn't talk about it. Why don't you talk about it?

[Laughter.]

Senator CARPER. We had this notion, silly notion that maybe we ought to have somebody who was pretty senior within EPA who would be part of the top dog for science and technology. It is an idea that was not warmly embraced by EPA and maybe by the Administration. We introduced it as legislation and not a whole lot has come of it, but I am still interested. I know Senator Voinovich is still interested in making sure that we are making decisions that are based on good science.

Let me just follow it up by asking you to talk a little bit more with us about CASAC, this Clean Air Scientific Advisory Committee. Just start out by saying, who are these people?

Mr. GRAY. CASAC is actually called for in the Clean Air Act, and its base is a seven member panel of experts with the requirement for one member to be a physician, one member to be from the National Academy of Sciences, and one member to be from a State pollution control Agency. Then it is filled in with nationally recog-

nized experts to help to provide advice to the Agency on ambient air quality standards.

On any specific standard, whether it is particulate matter, ozone, lead or something else, the base group of CASAC is augmented by topic-specific experts, again to help us in our evaluation, characterization, and presentation of the science.

Senator CARPER. Who selects or appoints these folks?

Mr. GRAY. They are selected by the Administrator.

Senator CARPER. What is the criteria used, just roughly?

Mr. GRAY. Roughly, it is relevant expertise and knowledge.

Senator CARPER. They serve a period of several years?

Mr. GRAY. I don't know the exact period. Yes, they serve for several years.

Senator CARPER. OK, thank you.

Why should EPA, why should the Congress, frankly why should anybody else heed the recommendations of the CASAC?

Mr. GRAY. Well, the EPA certainly values the advice of the CASAC. It is very, very important to us. The current proposal on National Ambient Air Quality Standards for particulate matter, for example, our proposal for a daily standard is something that reflects very clearly the advice of CASAC. Advice from CASAC, again, is one of the important factors that are weighed by the Administrator in making the sort judgments that are made.

Senator CARPER. What are the other factors?

Mr. GRAY. There are a range of other factors, comments from outside scientists, comments from others, the range of science that is presented to the Administrator. All of these are going to be before the Administrator. When we proposed our standards in every case, whether we were matching the recommendation of CASAC or not, we asked for comment. We asked people to advise us on levels that included everything that CASAC had recommended across a wide range of different potential standards. All of those comments and all of that information will be an important part of the factors that are weighed by the Administrator in making a final judgment.

Senator CARPER. Just to understand this, the current standards that exist are for annual, and there is a standard for daily exposures. If my life depended on it, I don't know that I could well explain what a microgram is, but I understand that the current PM standard on an annual basis is 15 micrograms per cubic meter. On a daily basis, it is 65.

Just explain that. As a toxicologist, I am glad that you aren't a lawyer for this question, but as a toxicologist and someone who studied some science himself, explain that so that the lay person can understand it.

Mr. GRAY. The numbers you are citing are the concentration of particles that are in the air. These are particles that are all around us right now. We can't see them. In our monitoring system, we suck air into a collector, put it on a filter, and measure it, and that is how we learn about how much is there. That information plays a role in our doing epidemiology, comparing the health effects in populations with higher levels of exposure to those with lower levels of exposure. It helps us to understand what happens when we give animals exposure to this, to help us understand the mechanisms by which adverse effects might occur.

So these standards, these numbers are simply ways of identifying a certain amount of this particulate matter that is in the air.

Senator CARPER. Again, drawing on your own expertise and training as a toxicologist, could you describe for us how these small, tiny pieces of matter, how they actually contribute to asthma, how they contribute to chronic bronchitis, how they contribute to heart disease or irregular heartbeat? How does it actually happen?

Mr. GRAY. That is one of the areas that we have learned much more about in the last 10 years, partially through research that has been funded by the Environmental Protection Agency. We know that at certain levels of exposure, for example, levels that are frankly higher than what we have in the air today, it can increase the thickness of atherosclerotic plaques.

Senator CARPER. Say that again?

Mr. GRAY. Atherosclerotic plaques, atherosclerosis, the stuff you worry about with cholesterol building up and making your arteries get small. That can happen in mice. They are sensitive mice. They are bred to be sensitive, but that can happen to them if they are exposed to high levels of particulate air pollution. It is one example of a study that has been done to help us understand how these particles may have their adverse effects.

Senator CARPER. My last question, Mr. Chairman, would be this, we have our current standard, and I think you said the annual standard is 15 micrograms. The CASAC had recommended I think going down to anywhere from 12 to 14. EPA chose to stay at 15. With respect to the daily standard, the current standard is 65. The CASAC had suggested going anywhere from 25 to 40, and you have come in at EPA at 35 micrograms.

You chose not to adopt or move toward the annual standard, and you chose, or adjusted the daily standard. You went to the high end of the CASAC recommendation. Could I ask why?

Mr. GRAY. Again, these are judgments on behalf of the Administrator and they are laid out very nicely in our proposed rule with the scientific reasoning behind each of the choices that were made. But the important thing, and I want to emphasize this again, I have said it a lot of times, we asked for comments about a range of other potential standards to get information from the outside community, from scientists, from the interested public about other potential levels, and that is all going to play a role in setting the final standard, and it will happen in September.

Senator CARPER. All right. Thanks very much.

Mr. Stephenson, sorry I didn't get to you, but another day. Thank you.

Senator VOINOVICH. I would like to thank both of you for your testimony before the committee. Mr. Gray, I want to thank you for your fine testimony here today, not an enviable position if you are getting shot at from both sides on this one. So maybe that is good. Thank you very much.

Our next panel will come forward: Dr. Roger McClellan who is the Advisor for Toxicology and Human Health Risk Analysis, and also is a CASAC, Clean Air Science Advisory Committee member; Dr. George Thurston from New York University; Dr. Anne Smith

from CRA International; and Mr. Dan Greenbaum from the Health Effects Institute, which is jointly funded by EPA and the industry.

I would like to urge the witnesses to limit their testimony to no more than 5 minutes. Your entire testimony has been inserted into the record. We really appreciate your being here today. We look forward to hearing what you have to say.

Dr. McClellan, we are going to start with you.

STATEMENT OF ROGER McCLELLAN, ADVISOR, TOXICOLOGY AND HUMAN HEALTH RISK ANALYSIS

Dr. MCCLELLAN. Thank you very much. Good morning, Mr. Chairman and members of the committee. I appreciate this invitation to present my views on EPA's current review of the national ambient air quality standard for particulate matter.

Since 1999, I have served as an advisor on issues related to air quality, drawing on my more than 45 years of experience in comparative medicine, toxicology, and aerosol science and risk analysis. In particular, my testimony draws on my experience serving on the Clean Air Scientific Advisory Committee, which I chaired from 1988 to 1992, and service on all of the CASAC PM panels from the late 1970's to the most recent PM panel, as we moved from consideration of total suspended particulates to PM_{10} to $PM_{2.5}$, and now consideration of a $PM_{10-2.5}$ standard.

I would like to make several points. First, I want to emphasize there is no scientific methodology which can determine a specific indicator, precise averaging time, numerical level, or statistical form that will be adequate to protect public health. The available scientific information can inform those decisions, however the Administrator must ultimately use policy judgments in making decisions on each of those four elements of the standard, drawing on an array of scientifically acceptable options.

Two, I personally find acceptable the Administrator's policy choices for the PM standard as were published in the Federal Register. Specifically, I find acceptable a proposal to reduce the 24-hour standard from 65 to 35 micrograms per cubic meter, with a 98th percentile form. I found it acceptable in terms of science for him to propose retention of the $PM_{2.5}$ annual standard at 15 micrograms per cubic meter. With reluctance, I concurred scientifically with the setting of a $PM_{10-2.5}$ indicator for the 24 hour averaging time concentration set at 70 micrograms per cubic meter, within a 98th percentile form.

I say with reluctance because the science base for that is extremely weak and uncertain. I would have preferred retention of the PM_{10} standard.

Third, it is important to recognize that although the criteria document is hundreds of pages in length and compiling the results of what we know in terms of the world of science about PM, at the end of the day the key information for setting the standards are the results of the epidemiological studies. That has been the basis for changes in standards from a total suspended particulates set in 1971, to the PM_{10} standard set in 1987, $PM_{2.5}$ in 1997, and now consideration of this new $PM_{10-2.5}$ standard.

So the current review focused on the $PM_{2.5}$ indicator. Now, some might have said, well, why didn't we look at sulfates or elemental

carbon or some particular chemical species? The fact of the matter is we are chained to the "regulatory compliance lamppost" in terms of monitoring data. You cannot conduct epidemiological studies unless you have the monitoring data. Our past obsession with monitoring that which is regulated has precluded the development of the richer science base that really need to consider these options in terms of speciation.

I am not optimistic that that is going to change. I can envision us sitting here 5 years, 7 years from now and discussing, well, why not for specific standards; why didn't we perhaps preclude or set outside of the standards on material, because it is very innocuous; or why didn't we focus on something in particular? In fact, we won't have the epidemiological data because I don't think we are developing adequate monitoring data.

Fourth, we can then turn to the question of toxicology. I am a toxicologist. I am intellectually married to the subject, if you will. We have exciting new methods at hand, but at the end of the day, I have to say that our toxicology methods are simply too blunt and yield results that can only be qualitatively extrapolated to the human population. I know of no scientific method for using the results of the toxicology studies with PM, including those conducted with specific chemical constituents, to develop quantitative numerical standards that are the core of the PM NAAQS.

Fifth, we have heard some discussion about the issue of uncertainty. I certainly, as a scientific colleague of Dr. Gray, am an enthusiast for trying to determine how we can bring our scientific information together and relate all of the uncertainties, so we can have better informed policy decisions.

I want to comment on one aspect of that, and that is the use of expert elicitation. That was covered in the GAO report. I served as one of those five experts on the pilot expert elicitation. I hesitate to say whether it is expert advice or expert opinion. It is a challenge when you are asked to present information. The answers that are given can be heavily influenced not only by your knowledge of the science, but your personal choices. All of us want quality life. Well, how do we separate that from our judgment on the science?

I am concerned about the use of that expert elicitation advice, and I certainly would urge the Administrator to use a high degree of caution in using that in the regulatory impact analysis and in making decisions on the standard.

Senator VOINOVICH. Dr. McClellan, could you finish up?

Dr. MCCLELLAN. I am going to just wrap up with that and emphasize that one of the challenges we have today is separating out people's views on the science versus their views on the science wrapped up with the policy and some desired outcome—the level of the standard. I think in some cases, as individuals and professional groups have weighed in, they are weighing in not just on the science, but the policy outcome they want.

I do think it is important to have a distinction between the science and policy choices. In my comments on the acceptability of the Administrator's choices, I viewed the science options laid out in the staff paper as acceptable, and he used his judgment in selecting from among those options. I think that was appropriate.

Thank you.

Senator VOINOVICH. Thank you very much for being here.
Dr. THURSTON.

STATEMENT OF GEORGE THURSTON, ASSOCIATE PROFESSOR, NEW YORK UNIVERSITY, SCHOOL OF MEDICINE, DEPARTMENT OF ENVIRONMENTAL MEDICINE

Dr. THURSTON. Good morning. I am George Thurston, a tenured Associate Professor of Environmental Medicine at the New York University School of Medicine, where my research involves the investigation of human health effects of air pollution.

I first wish to present for inclusion in the record letters from many major medical societies and public health groups, including the American Medical Association, the American Thoracic Society, and the American College of Chest Physicians, the American Public Health Association, the American Lung Association, the American Heart Association, the American Cancer Society, and more. These letters show the unprecedented support that exists for PM standards that are much tighter than EPA has proposed.

Especially note that they all agree that the science supports tightening annual standards to no more than 12 micrograms per meter cubed. If this were to be done, many thousands of premature deaths could be avoided each year.

I also present a copy of Dr. Rogene Henderson's letter from the EPA's Clean Air Science Committee, CASAC, urging that the Administrator comply with their recommendations, with CASAC's recommendations, to implement more stringent PM standards than now proposed by the EPA.

Now, in my written testimony, I have addressed three factors that need to be considered in the EPA's proposed revisions to the particulate matter air quality standards. First, I address the fact that we are now far more certain, as discussed earlier, of the adverse impacts and biological mechanisms of PM health effects. The uncertainties raised at the time of the initial setting of the $PM_{2.5}$ standard are now greatly reduced.

As outlined in figure one, if someone could put that up, from my testimony, the PM research funded since the setting of the last $PM_{2.5}$ standard has collectively shown the existence of numerous biological pathways capable of causing damage to the human heart, the lung, the nervous system and the circulatory system. This is consistent with the health impacts found by the PM epidemiology studies upon which the $PM_{2.5}$ standard was set.

This has greatly reduced scientific uncertainty associated with the mechanisms by which PM has such severe effects on human health.

Second, I documented reducing ambient PM levels can and do result in significant reductions in the mortality risk associated with this pollutant. Since the setting of the original $PM_{2.5}$ standard, more recent follow-up analyses of the landmark Harvard Six Cities and ACS studies have now considered longer records of time and have confirmed and expanded the conclusions from these two major studies.

As shown in figure two, an extended analysis of the Harvard Six City study through 1990 has now shown that reductions in long-term ambient PM pollution results in concomitant reductions in

health risks associated with PM. Large reductions in PM at four of the Harvard cities have resulted in likewise large reductions in the relative risk of mortality in those cities. We see S, Steubenville, where the pollution levels have come down, and so has the risk of mortality from that pollution. In Harriman, TN, the pollution levels have come down and the risk of mortality in that city has diminished as a result of the lower pollution. St. Louis, the L, has come down similarly, and Watertown, right, Boston, has similarly come down.

So the places where they have had improvements, we see that lower $PM_{2.5}$ lowers mortality. But I think it is important to say we still have a long way to go, and a lot of improvement yet to be made and benefits to be reaped from lowering the PM standard.

Finally, I show that the adverse health effects of PM air pollution extend below the $PM_{2.5}$ standard of 15. A recent NIOSH-funded extension of the ACS study, of which I was a principal investigator, strengthens the original conclusions of the ACS study, and it importantly now links increased risk of lung cancer to long-term exposure to PM, as shown here. As the pollution level goes up, the risks of lung cancer rise, as do all cause and cardiopulmonary.

As seen in this figure, the risks from $PM_{2.5}$ extend well below 15 micrograms per meter-cubed.

In conclusion, since it was the level of uncertainty about PM biological mechanisms and effects at concentrations lower than 15 micrograms per meter-cubed that limited the standard to that level in 1997, and, I point out, not some specific acceptable level of health risk from PM, and since new sound scientific studies have greatly reduced or resolved those uncertainties, then concern about the health of the public clearly indicates that the long-term $PM_{2.5}$ standard should now be reduced below 15, consistent with CASAC's advice.

And finally, I just want to point out that I was involved with the actual expert elicitation that has just been finished. It is finishing up under EPA. It is clear from the expert elicitation; I was shown the results of all the experts at the final meeting in New Orleans a few weeks ago. It is clear that the experts are, there is a consensus that, the risk from fine particle is much higher than previously thought, and that expert elicitation gives us an estimate of somewhere on the order of 1 percent decline in mortality per microgram per meter-cubed of fine particles.

So we are talking about a very large reduction in health risk, if you consider the fact that over two million people die every year in the United States, a reduction of 1 percent in that would be 20,000 deaths per year. So we are talking about many thousands of premature deaths that can be avoided by lowering this pollution.

I did want to respond to the question that you raised about asthma, if I have a second. Do I have time to respond to that?

Senator VOINOVICH. Why don't you bring it up in the question period.

Dr. THURSTON. OK, we will talk about it then.

Well, thank you for this opportunity to testify.

Senator VOINOVICH. Thank you.

Dr. SMITH.

STATEMENT OF ANNE SMITH, VICE PRESIDENT, CRA INTERNATIONAL

Dr. SMITH. Mr. Chairman, thank you for inviting me to participate in today's hearing. I am Dr. Anne Smith. I am a Vice President, CRA International.

I have been analyzing the risks and policy options associated with fine particle standards for over 10 years. The opinions I will present today are my own and not those of my company, CRA International.

When EPA set the first ever national ambient air quality standards for $PM_{2.5}$ back in 1997, in the face of substantial knowledge gaps at that time, it presumed that fine particles do have a causal relationship with public health. The law then required that standards be set for that $PM_{2.5}$ at a level that would protect the public health with an adequate margin of safety.

The courts ruled that the current standards they set did indeed provide that adequate margin of safety. EPA and the courts have also made it clear that that margin of safety does not eliminate estimated risk. Today, EPA is deciding whether new evidence accumulated since 1997 justifies tightening these standards. In both 1997 and now, EPA prepared a quantitative risk analysis using the available health studies. EPA says this is to help decide whether to tighten the standard. As I will show, it can help with this.

However, if you take the position that the $PM_{2.5}$ standard has to be tightened just because the risk analysis produces a body count at the prevailing standard, you will find yourself having pre-decided to tighten the standard in every future review cycle, even if the evidence never changes in the interim. This cannot be what Congress intended.

In thinking about whether to tighten the standard, the more appropriate question to ask is: Has anything changed in our knowledge that would undermine the Administrator's 1997 judgment regarding the adequacy of the margin of safety? Having a large number of new studies today, having successfully reanalyzed the studies we had originally, and even having some evidence of a still elusive biological mechanism, all fail to provide a justification to tighten the standard. They merely confirm that a standard is needed in the first place, which was the judgment made in 1997.

However, one could justify tightening the standard if the new evidence that I have just spoken of indicated that risks at the attainment level of the current standard have increased since 1997. To test this, I went back to my 1997 files to construct a comparison of EPA's estimates of mortality risks on a then and now sort of basis. I found that the estimates of mortality risk at the attainment level have actually fallen since they were first estimated in 1997.

Risks due to long-term mortality have fallen in every location. The risks due to daily exposure, which vary by city, have fallen in six of the eight cities that are in EPA's risk analysis. This new information suggests that the margin of safety provided by the current standards is actually greater than we originally thought.

What I have told you so far is based entirely on EPA's own point estimates of risk. However, it is worth looking more closely at those estimates and the basis for them. Every one of the new epidemio-

logical studies actually contains multiple alternative risk estimates. Sometimes there are dozens of them within a single paper. Which one is best? Which one should be used in the risk analysis? The Health Effects Institute has pointed out in the face of this issue that there is no gold standard for deciding this.

When I reviewed the full body of evidence in each paper that EPA used for its risk analysis, I went back to the original papers, I noticed that EPA had consistently selected the highest risk estimate or nearly the highest one from each of the papers. Therefore, EPA's risk estimates overstate what the full body of evidence supports today.

The evidence on fine particle risks has weakened in a number of other ways. Several of EPA's own point estimates today of the risk are not statistically significant. What this means is that behind the purported body count that comes out the risk analysis there is also actually a rather large probability of no health impact at all associated with that very same estimate. I found that none of the risk estimates for any of the eight cities in EPA's risk analysis remains statistically significant across all of the reasonable estimates in those papers.

This body of new evidence has also substantiated some of the concerns that were raised in 1997 that fine particles may be a scapegoat, while the real villain hides behind a veil of statistical uncertainty. The only clear trend in the new evidence has been that the estimated levels of risk that are being attributed to fine particles are lower than originally thought.

I would like to conclude by saying that we still have no idea of which constituent of $PM_{2.5}$, if any, is the culprit underlying this complex body of evidence. Even if the risk estimates are good, correct risk estimates, by regulating this generic mass of many compounds that is $PM_{2.5}$, we may gain little if any public health benefit.

Thank you for this opportunity to share my views on this important topic.

Senator VOINOVICH. Thank you, Dr. Smith.

Dr. GREENBAUM.

STATEMENT OF DANIEL GREENBAUM, PRESIDENT, HEALTH EFFECTS INSTITUTE

Mr. GREENBAUM. Thank you, Mr. Chairman. It is a pleasure to be back in front of the committee. I thank you for the opportunity to testify.

I am the President of the Health Effects Institute, which as you know is an independent research institute funded jointly by the EPA and industry to provide high quality, impartial science on the health effects of air pollution.

I am going to focus today on the science progress we have made since 1997, the most recent findings on the relationships between different ambient concentrations of PM and health, and key science needs going forward.

Since Congress identified the need for substantial enhanced research in 1997, much progress has been made. We know much more about the sources of fine particles and about personal exposure. We have conducted the first multi-city epidemiology studies

and analyzed and reanalyzed many of these studies, finding that in general the earlier studies were well done.

At the same time, there has been some evidence that the health effects we have seen in those earlier studies may in some cases be smaller. Unlike in 1997, we now have a number of toxicology studies that have begun to indicate potential biological mechanisms by which PM may cause health effects, although as always there is still much more to learn.

Among the most important questions addressed in recent years is whether exposure to PM has health effects at all levels, or whether there is a threshold below which no effects are expected. This question is central to setting the level of the NAAQS. This has been looked at in both short-term and long-term studies. Perhaps the most rigorous short-term study conducted since 1997 is the National Morbidity, Mortality and Air Pollution Study, which was funded by HEI and led by Johns Hopkins. That study examined daily changes in air pollution and health in the 90 largest U.S. cities.

As shown in figure one in my testimony, the study found that there appeared to be a linear relationship between mortality and air pollution for all causes of mortality and for deaths from heart and lung disease without an apparent threshold. Our review committee, which intensively peer-reviews all HEI research, advised caution in drawing conclusions from the apparent absence of a threshold, but noted that the reported associations in the study are at ambient concentrations well below the current U.S. daily standard.

There were two principal long-term studies in 1997, the Harvard Six Cities and the American Cancer Society study, and there have been some additional studies since, but attention has primarily focused on HEI's reanalysis of these two and on extended analysis in the ACS population.

For the reanalysis, we gained access to all underlying data in the studies and chose independent investigators who tested the original studies against a wide variety of alternative explanations. The investigators also conducted an analysis of the concentration response and figure two in my testimony presents the results, summarizing for each community the annual air pollution level and the risk of death due to heart and lung disease. As you can see, there is some scatter of effects. It is not all in a straight line, especially at the highest and lowest PM levels studied, but also an overall trend of increasing mortality risk with increases in pollution levels, starting at relatively low levels.

Following the reanalysis, the original investigators for the ACS study conducted an extended analysis of the data, which Dr. Thurston has mentioned and participated in, and found similar results.

While we have made much progress, there continue to be, as there always are in science, important questions. Two key areas are: First, we need continuous improvement in the statistics used in epidemiology to better test the sensitivity of the results, to quantify the uncertainty, and to communicate both the results and the uncertainties clearly.

Second, no other question will have as much impact on future regulations than determining whether some components of the

complex mixture of PM are more toxic than other components. Ultimately, this data will be essential to ensuring that regulations are targeted at reducing those emissions which will have the most public health benefit at the least cost.

This has also become important in light of the current proposal for a PM NAAQS for coarse particles, which is proposed to exclude certain particles from regulation. There have been some individual city studies of this question, but no systematic national effort to compare results from both epidemiology and toxicology. To fill that gap in time to inform a next round of PM NAAQS review, HEI has launched, with support from both EPA and multiple industries, a set of systematic multidisciplinary national studies.

As indicated in both the NRC review of PM research priorities and in today's GAO report, these PM component studies will be central to ensuring that future PM actions are the most effective possible.

I might add that while I obviously have the utmost respect for Dr. McClellan and take his skepticism that we can answer this question as a true challenge, as a long-time Boston Red Sox fan and a hopeless optimist, I enter this challenge with the hope that we can, if we all put our minds to it, answer this question of which components much better 5 or 6 years from now than we can today.

Thank you for this opportunity to testify. I would be pleased to answer any questions you might have.

Senator VOINOVICH. I want to thank all the witnesses for your testimony this morning.

Before I start the questioning, Chairman Inhofe asked that I insert testimony for the record from Dr. Borack on the issue of coarse particulate matter. Without objection, we will do that.

[The referenced Testimony can be found on pages 171-187.]

Senator VOINOVICH. Mr. Greenbaum, as you know, there is an ongoing debate about whether EPA must lower the annual standard. We talked about it last week, and this week we are trying to get some of you that are smarter than we are in the scientific area to give us your best thoughts.

Your opinion is invaluable in this debate as an entity that sits squarely in the middle. You are funded by both industry and the EPA. Clearly, the statute says the standard shall be set, "in the judgment of the Administrator." In your opinion, is it reasonable from a scientific and health perspective for the Administrator to retain the annual standard at 15 and not lower it?

Mr. GREENBAUM. Well, Mr. Chairman, I appreciate that that is the $64 million question at this hearing. I tried to present the best view of the science that we at HEI can give you on that question. While the dose response or the relationships going down to the lowest levels are very clear for why we are tightening the daily standard, they are strong, but not as clear, for the long-term studies, as shown in the data I presented.

There is evidence with cities showing reduced effects as you go down below 15 micrograms per cubic meter. I think it is exactly in that area where there is some certainty about these results, but where the Administrator has to make a policy judgment about whether that is certain enough to require additional actions. As Dr.

McClellan said, there is really no scientific method even that HEI has that could set that.

I should add that we at HEI, since our inception, have never taken a position on a specific standard because of the clear concern that such a position would be viewed as therefore biasing any of our future science. So we don't take positions on this. We don't advocate. The science is definitely stronger on the concentration.

Senator VOINOVICH. The question is, is it reasonable?

Mr. GREENBAUM. I think I am going to have to leave that to the Administrator to make that judgment. I think that there is evidence that we didn't have in 1997 of effects still continuing below 15. But whether that is enough evidence to make a call for a reduction in the standard is a public health policy judgment, not a scientific judgment.

Senator VOINOVICH. Dr. Smith, what is your opinion?

Dr. SMITH. The way to think about setting the annual standard is to look at the chronic health studies. The chronic health studies have been the ones that have been found to have FE.

Senator VOINOVICH. What studies again?

Dr. SMITH. I am sorry. Chronic health studies, which are studies of long-term exposure to $PM_{2.5}$ so they are more relevant to measures like the annual standard and annual average, rather than to a day to day peak, which is the daily standard. So the studies that look at the long-term exposure of $PM_{2.5}$ with mortality are the ones that are in question for whether we need to tighten the annual standard. Those studies are some of the most difficult ones to properly control for in statistical methods.

What we found in the reanalysis that Dr. Greenbaum's organization performed is that in fact when reanalyzed with alternative methods of control, the estimate gets more and more insignificant as additional factors are brought into play such as SO_2. SO_2 when added into the analysis causes the $PM_{2.5}$ health effect to become insignificant, issues that are complex statistical issues, and I will use a technical term, spatial autocorrelation, are problems if you have those in the analysis. They were found in the health effects studies for long-term standards and when controlled for, the effect becomes statistically insignificant, again meaning a very large probability that there may be no effect at all.

So the evidence has weakened since the standard was set at 15 micrograms per cubic meter based on that data set. I will also say, when that standard was set, there was evidence of effects below 15 in that data set. That hasn't changed. That is not new information. So while the analyses have held up to reanalysis and been confirmed that you can find this sort of correlation in the long-term studies, they are much weaker in terms of how well you can make a causal interpretation of them.

Another very important finding that came out of the Health Effects Institute's reanalysis is that in fact if you break the population that the data, if you break the cohort that is being studied into three groups, different educational levels, and you look at the group that has even 1 year of education beyond high school, which is what good deal of our population has, there is no effect at all. It is gone. It is not in that analysis.

So the effects are very dependent, the effects that are being purported and attributed to $PM_{2.5}$ out of that study are now known to be very dependent on whether a person has had any education beyond high school or not. That relationship is found in all the studies.

Senator VOINOVICH. What does that mean?

Dr. SMITH. It suggests that there is a missing variable in the equation, basically. We know that education does not in itself make somebody more or less susceptible to exposures to fine particles, but there may be something.

Senator VOINOVICH. Does this mean that the people that are 1 year in high school live out in the suburbs and others live in the urban area and are more impacted by this?

Dr. SMITH. It may have something to do with the poor evidence on what they are being exposed to. It may be something about lifestyle that actually creates risk for them that the $PM_{2.5}$ is picking up on, but isn't really exacerbating. It is just serving as another thing, a scapegoat.

Senator VOINOVICH. Let me ask you something. If I am a smoker, and please forgive me if these are elementary question, but if I am a smoker, am I more subject to fine particle PM than if I am a nonsmoker?

Dr. SMITH. I actually am not quite sure how that relationship works out in the study, but they have controlled for smoking in the relationship. Certainly, smoking is related to educational level, but smoking was controlled for when they did this analysis of whether there was an effect at different educational levels. So the correlation between smoking and educational level is not the education for what is going on there. It means there is something totally different at play.

Until you find out what that problem is, through more study, more research, and it can be done, until you find out what that problem is that explains this unexpected and bizarre result, you don't have an unbiased estimate of what the risk is. It could be larger or it could vanish, the $PM_{2.5}$ risk, that is.

Senator VOINOVICH. OK. In terms of the particulate matter, again, we are trying to reduce NO_X and SO_X, mercury. When they do the analysis of the particulate matter, they just segregate that out and you don't take into consideration the impact that NO_X or SO_X. Or does particulate matter have something to do with NO_X and SO_X?

Dr. SMITH. The results that are being used in the risk analysis do not account for SO_X or NO_X or any of the other pollutants and whether they may have an explanatory role, too. As I said a minute ago.

Senator VOINOVICH. In other words, when you are doing the, I always have a tough time with that word, epidemiologist do the studies, OK, the fact is that they are just looking, they are trying to figure out just what the particulate matter is. They don't take into consideration that I am exposed as a person to both NO_X, SO_X, particulate matter, mercury and the rest of it, and somehow they are able to pull out and say these particulate matters are the things that are really contributing to morbidity.

Dr. SMITH. As I was saying, the mortality, as I was saying, the estimates that are being used right now to estimate risk from $PM_{2.5}$ only consider $PM_{2.5}$. However, the studies in the many, many other results that are in these papers look at other formulations, other methods of doing the estimate that put the other pollutants in to the model as well and try to understand which pollutant is it. Does one of them explain the effect that we are seeing for $PM_{2.5}$ when that is the only one in the model? Is there another pollutant that could explain it better, when we put them both in the model?

What is found in study after study after study when this is done is that the $PM_{2.5}$ effect goes away, becomes statistically insignificant is what I mean. The other pollutant that is in the model that is being explained by the analysis at the same time retains its statistical significance. I looked at all the $PM_{2.5}$ short-term studies that I could find that did this analysis, this type of two pollutant analysis. I found that the vast majority of them had the $PM_{2.5}$ effect that was statistically significant if it was the only one in the model, become statistically insignificant when one of the gaseous pollutants, and these included ozone, SO_2, NO_X, and carbon monoxide. The same thing is going on in the chronic studies for SO_2.

Senator VOINOVICH. To get back to the original question. If the Administrator, is it reasonable from a scientific and health perspective for him to retain the annual standard at 15 and not lower it? Is it reasonable?

Dr. SMITH. Given the basis for the standard of 15, which was the same chronic studies that I have just described to you, but before we studied them and discovered some of the underlying uncertainties in them, and given that the risks are lower now based on the new evidence using their point estimates, it seems quite reasonable to say that we haven't learned anything that says that that standard has a smaller margin of safety than it had when the standard was set.

Senator VOINOVICH. So you would say that if he did it, it would be reasonable.

Dr. SMITH. That could be reasonable.

Senator VOINOVICH. May I ask another question? In terms of sulfur and NO_X, in terms of public health, and particulate matter, which is the worst?

Dr. SMITH. Between sulfur and NO_X SO_2 and NO_X

Senator VOINOVICH. Sulfur dioxide or NO_X, if you were able to reduce those substantially, in terms of public health, which one of those is the worst?

Dr. SMITH. I really cannot say.

Senator VOINOVICH. I would be interested in all of you.

Dr. SMITH. I just don't know. I have not actually done a relative analysis of SO_2 and NO_X.

Senator VOINOVICH. Well, has anybody done an analysis at the table? Dr. Thurston, you have been around a long time. You have testified a lot of times before this committee. Do you have an opinion?

Dr. THURSTON. Well, yes, I would like to respond to a few things that were just said, including that.

Mr. GREENBAUM. Senator, I realize you are going to go through everybody, but I would like, since it was HEI who conducted the

reanalysis I would like to have the opportunity to clarify for the record what we did conclude.

Senator VOINOVICH. Get your mike there, please. Go ahead.

Mr. GREENBAUM. I'm sorry.

Senator VOINOVICH. OK, go ahead.

Mr. GREENBAUM. I just want to, and I don't mean to take away from Dr. Thurston, but I did want to clarify what the HEI reanalysis of the long-term studies in effect did and didn't find, because there has been some degree of confusion just thrown into it unfortunately by Dr. Smith's comments.

First of all, I think everybody can in this process pick their favorite study and their favorite result from the study and try to emphasize that, so they can emphasize the one that has multiple pollutants in it and say there is no effect, or they might emphasize the one that has no other pollutants but PM in it and say there is a big effect of PM.

Our Review Committee, in our review of the reanalysis—this very intensive look these studies—had to look across all of the analyses to figure out which ones were the right ones, which ones were the wrong ones, which ones were statistically stronger or weaker. While, of course, that group of scientists identified continuing questions for those studies, we did find as strength of the evidence that the effects of PM were robust to a number of analyses that were done.

We also found for the first time a strong effect of sulfur dioxide in that, although our committee, and this is in partial answer to your question, did not think that sulfur dioxide itself could be attributed to doing that, based on toxicology information, but that it might be a marker for something else in the air that would travel with it, but we are just not measuring that, and therefore we don't have information about that other substance.

The reanalysis definitely found, after test after test after test, stronger results from those studies, with some continuing questions. We certainly had that.

Second, on the question of education, and how education modifies the results, certainly there was a stronger result shown for those with the least education in terms of increased mortality. We do largely view educational levels as one marker for poverty or socioeconomic status. We know that poverty and socioeconomic status themselves can have effects on health. People can have less good medical care, less good nutrition, and a number of other things that shorten their lives. We know that and our reviewers certainly understood that as well.

In that analysis, in the reanalysis, however, when you looked at heart and lung disease deaths, which is not what Dr. Smith put in her testimony, you still see some effect in everybody else in the population. That effect was marginally statistically significant, but still there. Our investigators, our reviewers felt two things, that: One, that there was a continued effect at these different levels, with the strongest effect being that those with the least education and probably least socioeconomic status, and that might be because of higher exposures, less access to air conditioning, and a number of other things that would increase the effect.

Two, they also did note that no study is perfect, so there probably is still, somewhere in there, some continued confounding where the results are showing an effect of being poor, but they didn't suggest that that explained the whole effect. I just wanted to clarify that.

And then they did test all of the pollutants in the reanalysis, including nitrogen oxides and sulfur dioxide and ozone. Sulfur dioxide was the only one that showed effects and had any effect on the PM results. As I said earlier, they thought that was important to understand. They understood that we need to do more work on that. They weren't sure that it was the sulfur dioxide. In fact, the sulfur dioxide might have been a marker for other types of particles that are coming along with the sulfur dioxide, not the gases themselves. There could, for example, be metals that come out of the same sources.

So I thank you for the opportunity. I just wanted to clarify what our reanalysis did say, and obviously the full report was actually presented to you at an earlier hearing.

Thanks.

Senator VOINOVICH. Dr. Thurston.

Dr. THURSTON. Yes, thank you.

Before we start out, you were noting that Dr. Greenbaum is between Government and industry. I just want to point out that I represent, I think, a disinterested party. I am a tenured professor at NYU. My salary is paid by NYU. I don't do consulting for the vested interests involved here. So I feel that my testimony should be viewed as a disinterested party, not an uninterested party, but a disinterested party.

I have to agree with Dr. Greenbaum's discussion of the SO_2 and the HEI reanalysis. SO_2, it is clearly stated. You know, I think that unfortunately what Dr. Smith has done here is exactly what she accuses EPA of having done. She has cherry-picked certain results that support her position and unfortunately hasn't looked in a balanced unbiased way at this question.

Senator VOINOVICH. Can I ask you something?

Dr. THURSTON. Yes.

Senator VOINOVICH. What is it that contributes to the particles? You have coarse particles, you have fine particles. We are becoming more concerned about fine particles. I know I have been at the University of Cincinnati, the Children's Hospital down there. They are doing a study of diesel fumes on urban kids——

Dr. THURSTON. Yes, I have done that myself.

Senator VOINOVICH [continuing]. The development and so forth. But we are talking about the things that cause the particles. Is it basically emissions from automobiles? What is it? What is the cause of this?

Dr. THURSTON. On that question, you made a good point, you know, that these are oftentimes markers. I think the SO_2 being a marker of largely power plant pollution in the United States; NO_X being largely a marker of traffic. It is very likely, actually, that that SO_2 in the ACS study, because I have done some of this analysis, of course, as being PI of the NIH-funded portion of that, that the SO_2 is a marker for power plant pollution, and that contribution to mortality.

It depends on the health outcome you are looking at and where you are.

Senator VOINOVICH. The particulate matter we are talking about, you know, what is the biggest source of particulate matter?

Dr. THURSTON. Well, there are different types, and I think what we are wrestling with is: Which effects are caused by which types? But I think it is safe to say if you lower the fine particle levels, you are going to better protect health. So the standard is necessary. It is appropriate. As we learn more, we may be able to refine that further.

Senator VOINOVICH. Just for example, we are trying to make things better, and we are going to set a standard. Well, somebody is going to have to meet a standard, but if you were going to meet the standard, do you do that by reducing diesel emissions? Or what is it that is going to have the biggest impact on, if you assume what you are saying, that fine particle matters are bad and if you set it at 12, everybody's going to live longer than if you have it at 15, what is it that you have to do in order to make sure that you don't expose people to these things?

Dr. THURSTON. Well, I think that is going to vary depending on where you are. If you are in Los Angeles, I think traffic would be a good thing to try, the pollution from traffic. If you are in the Northeastern United States, power plants are a big problem there, but not so much in Los Angeles.

So I think that the States will implement controls on the pollution problems that are the largest. I think as we go on, I mean, this is going to take us 20 years before we are finished with this process, in reality, of meeting the standard that is set. So as we go through it in that time, we should be able to provide the kind of research that HEI is funding, that EPA is funding. In 5 years, I think we will be able to give you a better answer. As the States move to the implementation phase, they will have that information about the sources.

Senator VOINOVICH. But the answer that they come up with is that at 12 it would have a major impact on the economy of this country and the environment in various places. There is just no question about it.

Dr. THURSTON. But it would have major benefits, health benefits of lowering it to 12. I agree with that. The past has shown that environmental controls are not a big problem economically. We have been able to. We have other problems with economics, as you are well aware.

Senator VOINOVICH. Dr. Thurston, let me tell you something. I am a former Governor of Ohio, OK. The first thing I did was try to get my counties into compliance with the current ambient air standards because my businesses told me they weren't going to expand, and we knew from an economic development point of view that those counties that weren't in attainment wouldn't even get smelled by businesses that we were trying to bring to our State.

Today, with the new rule, we have a number of counties throughout the United States that are not compliant with particulate matter. They have this stamp on them: don't expand, cost you more, don't go there, cost you more. If we reduce the standard from 15 to 12, and there are going to be a whole lot more of those through-

out the country, it will have an impact on the economy of those respective communities.

Now, that is not supposed to even be paid attention to, so they can't do it, but it seems to me that we have a lot of speculation going on whether it should be 15 or 12 or 14, that we ought to be pretty sure, pretty certain about some of the conclusions that we come to.

I have a problem, that I am just wondering about, you know, the budget, the monitoring budget, the $17 million. Senator Carper and I have been trying to get them to have an Assistant Administrator for Science in the EPA. Does the EPA have the brains and the people to get the job done?

Dr. THURSTON. I can't answer that one. But you know, I think the last time we spoke, we both agreed that we as a country need to set a standard for the rest of the world, and we need to develop the technology to meet these standards and then sell it to the rest of the world.

Look at China. The pollution problems there are huge. That is a huge market for the United States. If we develop the control technology here first, if we confront this problem first, we can then turn lemons into lemonade, if you want to view it that way. We can be first to clean the air, and sell that to the rest of the world. We agreed on that, as I recall, the last time. I think it is time we got on with that process.

Now, to get back to the issues, this education question. Being one of the authors of this, we have looked into this. I agree with Mr. Greenbaum on this. I just would like to point out, one of the things we have looked at is the migration question, and that is that people of lower income tend to not move out of their MSA. They are not highly mobile. We looked at the data and they tend to stay exactly where they are year after year after year.

Whereas higher educated people are much more mobile, and that makes it so that the exposure estimates that we are using in our studies are less reliable and therefore, some of them move to higher exposure areas, some move to lower, so that we are going to have greater difficulty finding an association in those places.

That leads us to believe that the estimates of the lower income people are probably more accurate and yes, the higher income people, the higher educated people are having the effects, but they don't show up in a study because the studies' estimates of their exposures are not as good as for the less educated people.

So I think what that says is that what we have reported is the average of all three. Probably the lower income people are giving us the most accurate estimates, which means they are much higher than we thought, and that is part of the equation why, in the expert elicitation, the various experts, a dozen experts, have come up with estimates much higher than the ACS study because they have adjusted for this factor and others that have come.

So when you resolve uncertainties, we are not going to lower estimates of the pollution. We are resolving uncertainties and figuring out what is going on, and actually, the impacts are going up in terms of the long-term exposure which is, you know, that is what most of the deaths are associated with, the long-term exposure to

this pollutant, which is pervasive year after year, day after day, for every American.

Senator VOINOVICH. Thank you.

Senator Carper, I didn't think you were coming back. I was taking everybody's questioning time.

I think in fairness to Dr. McClellan, we have less than a minute, because Senator Carper is here.

Dr. MCCLELLAN. I would like to respond to your question in terms of lowering the annual standard.

Senator VOINOVICH. The question was reasonable, would it be reasonable?

Dr. MCCLELLAN. Yes. I am on record as stating that I think that the staff paper laid out the science. It had a range of 12 to 15 mg/m^3. I felt that was the appropriate expression of the science, and, thus, the Administrator's policy judgment to go with 15 mg/m^3 I thought was appropriate.

I did not think, as a member of CASAC, that it was appropriate to narrow the range of 12 to 15 down to 13 to 14. In fact, I thought that was just one step away from saying, this is the standard that should be set. I thought that overstepped the bounds.

I would like to use one of Dr. Thurston's graphs, if I could, to point out some of the difficulties here. This is the Harvard Six Cities study, started back in 1979 which I view as a platnium study. The late Dr. Ben Ferris was the guiding light that pulled together people in teams of six different communities. One of those happens to be in your State, Steubenville. OH. It started as an extensive study I think of about 8,000 people over time.

Now, here are the results from this. I think it is important to look at the graph. Across the bottom we have $PM_{2.5}$ micrograms per cubic meter, zero to 30. Now, we have a single number there on that. Now, you will note that each of the cities has changed over time. Steubenville was originally up close to 30 and now it is down just above 20. You can't express in this graph what it was in 1950? What was it in 1940? Most of the deaths that are expressed in here are of the elderly. We know that. In the United States, people live long lives. So in any expression of death, they are going to be the individuals being counted.

What we are not able to show on this graph is what these people were exposed to in those earlier years before we had the monitoring? We know that that was substantially higher, so we haven't captured that value in this data. What I am willing to say, looking at long-term data, is those values must have been substantially higher.

Now, we look over here at the mortality relative risk of mortality related to PM exposive. I want to emphasize, we sometimes talk about excess deaths. It is important to recognize that when we conduct epidemiological studies we do not create extra deaths. What we do is try to take the deaths that have occurred and tease out what they may have been associated with.

In terms of cardiopulmonary deaths, we know the biggest risk factor is cigarette smoking. If you go to the data of Engstrom, out of LA, his relative risk factors for the all cause mortality for smokers, about a pack a day, was 2.0. That would be way the heck up

there on this graph. A value of 1.0 means there are no deaths assignable to PM.

So one of the things we do know if we look in terms of the baseline for this relative risk, death rates for cardiopulmonary deaths have been going down. For lung cancer, we have seen for the first time a decrease in lung cancer mortality. Why, because people quit smoking?

So the data that Dr. Smith related to, I suspect, if I were able to examine her calculations, we would find that in all of the eight cities, there were improvements, and so the base for that relative risk factor is going down resulting in fewer calculated deaths related to PM in the air.

What I am saying is when you look at all of this data, I think the Administrator was fully appropriate in his policy judgment to continue at 15 micrograms per cubic meter, and I think when that is ultimately met across the country, we will have I think some small improvement in total health.

Senator VOINOVICH. I thank you very much for that. I will turn it over to Senator Carper. I just couldn't help, and I think you will understand this, I will never forget campaigning for Governor in 1990 in Steubenville, OH. I ran into a woman who was an immigrant, and we were talking about the air, and she said, "Mr. Voinovich, I put the sheets out in the old days and there would be black stuff on it. Now I put it out, no black stuff. The sheets are clean, but nobody is working."

And that was it. We didn't get into the health or anything else, but the fact of the matter was that things were better for her environment, but the thing that she was concerned about is that nobody was working and wanted me to do something about bringing jobs to Steubenville, OH.

Senator CARPER. That was actually a pretty good segue, because I have just come from a hearing in the Banking Committee which is going on downstairs. Every 6 months, the Chairman of the Federal Reserve comes and testifies to brief the House and the Senate on monetary policy. He was just sharing with us how strong our GDP growth is, 5.6 percent I guess for the last quarter, how many new jobs are created thus far this year, and large growth in Federal revenues and that sort of thing.

But the progress, the economic growth is uneven across the country. Frankly, the growth in incomes is uneven across our citizenry, as we know.

I apologize for missing your testimony. Whenever the Chairman of the Federal Reserve comes and testifies, he doesn't do it often, I try to be there. I like to kid and say he won't go forward until I am there, but that is not really the case. But I wanted to be there to be able to offer a statement or two and maybe ask a question of him.

I appreciate the fact that you are here. We appreciate your testimony, your input, and your willingness to respond to our questions. I have a couple of specific questions of Dr. Thurston. Before I do, I have sort of an unwritten rule that whenever one witness uses the charts of another witness to make a point, we always give the owner of the charts the opportunity to have the last word. Do you

have any comment or any response you want to make to Dr. McClellan's comments?

Dr. THURSTON. Yes, I do. I thank you for that opportunity.

Well, you know, I think that his reference to, well, first of all, his references to cigarette smoking, these studies have been controlled for cigarette smoking.

Senator CARPER. Say that again?

Dr. THURSTON. Have been controlled for, this study that is represented here has been controlled for cigarette smoking.

Senator CARPER. OK.

Dr. THURSTON. OK. So these reductions in relative risk have nothing at all to do with cigarette smoking and the fact that people are smoking less. This has to do with the reductions in air pollution after controlling for all these other factors that they studied. This is a cohort study where they had individual information about each of the people in the study.

The other thing he is talking about, well, exposures long ago that could be responsible for these effects. There is recent research that indicates that it is really the exposure in the last 5, at most 10 years of your life, or up until the time of death that are the most important. Even if you look at cigarette smoking, when someone quits cigarette smoking, the Surgeon General's report points out, within 10 years their mortality risk is the same as the general public. It is similar with air pollution. The benefits are yielded very quickly, actually, after the exposure is reduced. We see that in this study and we see it in others.

We are looking at the ACS study, looking at these windows of exposures, and we will be publishing on that as well. But I know there are papers out there, like Kunsley's recent paper pointing this out. So that is really my response to his comments.

Senator CARPER. Good. Well, thank for sharing that.

Perhaps a couple of specific questions of Dr. Thurston. Dr. Smith and Mr. Greenbaum, I missed your testimony, as you know. What I am going to ask you to do is, take no more than a minute, but just give me my takeaways. It is impossible for us to remember everything you said or say, but just, if we remember nothing else, of a couple of key points that you made, what should we take away from here?

Dr. SMITH. Just a minute.

I think the key takeaway is that if you want to understand whether to tighten the standard, rather than whether we need the standard that we have, then you need to look to the question of what is happening to the margin of safety that was deemed acceptable when that standard was set, and using the new evidence that we have today is that the margin of safety that is associated with the current standard has, if anything, grown, not narrowed.

So while there is better information across the board, there does seem to be confirmation that there is some subtle effect going on with air pollution, that was assumed at the time the standard was set, and the standard at that time was set so that it provided a margin of safety, and that margin of safety remains today even in the face of all of this new evidence.

Senator CARPER. Thank you.

Mr. GREENBAUM.

Mr. GREENBAUM. Sure. Just three things. First, we have made tremendous progress since 1997 in understanding a whole series of questions we had then. In fact, they are not even on the table now in the same way because of research that was chartered by Congress and then put into place over the last 6 to 7 years to answer those questions.

Second, one part of that research has been to look at the so-called concentration response, what happens at higher and lower levels of pollution? Do we see more effects, less effects? Is there a point below which we don't see any effects? And there, we have seen, 0 with some uncertainty at both ends of the range, generally increasing effects with increasing levels of pollution, a generally linear relationship between those two.

At the same time, and the third point, looking forward, there are still, as science always has, important continuing questions, probably most importantly, the one around understanding whether there is a different toxicity of different components of PM, really different sources of PM, to guide future regulatory decisions.

Senator CARPER. Good, thanks. My thanks to both of you.

Dr. Thurston, a couple of questions, if I could. Do you believe that there is certain, I would underline the word certain, scientific evidence to justify lowering the annual standard?

Dr. THURSTON. Yes, I certainly do. The bulk of the evidence, as EPA has presented it, shows that we need to tighten these standards, that there are health effects below the present standards and that they need to be tightened.

Senator CARPER. OK. I think, and I didn't hear this issue, but Dr. Smith I believe you may have suggested that the risk of fine particles has actually decreased in your testimony. I would ask of Dr. Thurston if you want to comment on that. I don't want to mischaracterize what she said, but if I have that right, would you just comment on it?

Dr. THURSTON. Well, as I said in my testimony, I believe that, and I think the evidence supports, that the uncertainties associated with our estimates have actually decreased. We are more certain of the effects, the biological mechanism, and the size of the effects. So uncertainty has been diminished.

I want to clarify the distinction between, I guess, uncertainty and doubt. There is no doubt that air pollution is causing these effects, and there is no doubt that lowering these pollution levels will reap huge health benefits. There is uncertainty exactly how much benefit for how much pollution. So that is uncertainty of the estimate. It could be higher. It could be lower. In the case of the long term, the scientific consensus is moving that the health effects of $PM_{2.5}$ long-term exposures are actually higher than we previously thought.

So there is uncertainty around the estimates, but there is really no doubt here.

Senator CARPER. All right. My thanks to each of you. I am glad you were still here when I got back. Again, I appreciate very much your testimony and responding to our questions. Thank you.

Senator VOINOVICH. Thank you much. I really believe that I have gotten some more clarity here, at least in terms of people's dif-

ferences in terms of this standard. Hopefully, this will help the Administrator make the right decision.

Thank you very, very much. The hearing is adjourned.

[Whereupon, at 11:30 a.m. the committee was adjourned, to reconvene at the call of the Chair.]

[Additional statements submitted for the record follows.]

STATEMENT OF HON. JOSEPH LIEBERMAN, U.S. SENATOR FROM THE STATE OF CONNECTICUT

Thank you, Mr. Chairman.

In my view, EPA's most fundamental responsibility under the Clean Air Act is to tell Americans truthfully whether the concentrations of pollution in the air they breathe are at levels that endanger their health. If EPA knows that particulate matter hurts people at lower concentrations than those reflected in the agency's existing air-quality standards, then I believe the agency has a legal and a moral responsibility to tighten the standards.

Following an exhaustive review of peer-reviewed studies on the subject, the scientists, doctors, and public servants on the congressionally chartered Clean Air Science Advisory Committee have told EPA that air-borne particulate matter is triggering large numbers of asthma attacks, heart attacks, and premature deaths in many areas of the country that meet EPA's existing air quality standards for particulate matter. That leads me directly to the conclusion that EPA must make those standards more stringent. An alternative risk analysis requested by the White House Office of Information and Regulatory Affairs has just come to the same conclusion.

Unfortunately, EPA has proposed to not lower the annual particulate standard at all, and to lower the daily standard to a level that remains above the limit that the Science Advisory Committee has identified as necessary to protect public health.

I do not believe that EPA can justify this disregard for the Science Advisory Committee's recommendations. Skeptics enjoyed representation on the Committee and ample opportunity to press their views. To me, it makes no sense to disregard the Committee's conclusions based on complaints that were not sufficiently compelling to convince that expert body. Here I note that only two of the twenty-two members of the Committee's panel on particulate matter dissented from the panel's conclusions, and that all seven members of the committee agreed with the panel's majority.

Having reviewed the statements and testimony delivered at last week's hearing, it appears to me that the impetus behind the calls for EPA to disregard the Science Advisory Committee's recommendations is not dissatisfaction with the scientific work of the Committee, but rather concern over the cost of bringing air quality into line with more stringent standards.

Clearly, achieving further reductions in particulate-forming emissions will cost money. That is why the Clean Air Act's system for implementing the health-based air-quality standards includes, at nearly every turn, generous regard for what is practicable and what is not. There is no need, then, to flout the Act by infecting the standard-setting process with considerations of implementation costs. I would point out, moreover, that by any reasonable measure, the economic benefit of the lives saved and illnesses averted by bringing particulate levels down to the levels recommended by the Science Advisory Committee would vastly outweigh the economic costs of the added pollution controls.

The concentrations of particulate matter persisting in many parts of the country cause more than 45,000 premature deaths every year. The problem is too grave and too large to be concealed. We can solve this problem, and the first step is to level with the American people. That is why I urge EPA to set the revised particulate matter standards at the levels that the Science Advisory Committee has determined necessary to protect public health. Thank you, Mr. Chairman.

STATEMENT OF GEORGE GRAY, ASSISTANT ADMINISTRATOR FOR RESEARCH AND DEVELOPMENT, U.S. ENVIRONMENTAL PROTECTION AGENCY

Good morning, my name is George Gray, and I am the Assistant Administrator for the Office of Research and Development in EPA. I wish to thank you for the opportunity to discuss the EPA's evaluation of the scientific evidence for potential health effects of airborne particulate matter (PM). Last week the Subcommittee on Clean Air, Climate Change and Nuclear Safety heard from my colleague, William

L. Wehrum, the Acting Assistant Administrator for Air and Radiation, on the EPA's review of the national ambient air quality standards (NAAQS) for PM. During that testimony he explained the crucial role of science in helping to inform decisions about the National Ambient Air Quality Standards.

Today, I would like to talk with you in more detail about this science: how it is prioritized and developed, and how it is synthesized and integrated. As Assistant Administrator for EPA's Office of Research and Development (ORD), I am responsible both for the development of new scientific information targeted to address critical Agency research needs and for the evaluation, synthesis, and integration of the world's peer-reviewed science literature into a document that informs EPA decisionmakers. As we characterize the current state of our collective scientific knowledge, we are careful to point out the strengths and weaknesses of this large body of information, so that informed decisions can be made. It is clear that the scientists and staff of ORD play a crucial role in the development and evaluation of the world's scientific information to inform the review of National Ambient Air Quality Standards.

We all agree that environmental protection efforts must be based on high quality science. High quality science includes both the conduct of research—in the laboratory and in the field—and the careful evaluation of that body of research to inform policy making. High quality research is focused appropriately on generating new knowledge that addresses complex scientific issues and helps reduce important scientific uncertainties. It is carefully planned, well conducted, and thoroughly peer reviewed by independent scientific experts. The careful and balanced characterization of the body of knowledge created by high quality science requires an open process, interaction with appropriate subject matter experts, and serious consideration of the ways in which the results are communicated to decision makers. To me, an important component of high quality science is the characterization of the uncertainties related to individual studies and, more generally, the characterization of the weight of the scientific evidence.

First, let me discuss EPA's efforts to develop new and relevant science on particulate matter. The Agency has a longstanding and strong program to develop and use new scientific knowledge on the health effects of airborne PM. After the last review of the PM NAAQS in 1997, EPA embarked on a very ambitious research effort to advance our knowledge and address important uncertainties in the science related to PM. Congress requested that we sponsor the National Academies of Science (NAS) to provide us advice. The NAS Committee on Research Priorities for Airborne Particulate Matter in the National Research Council completed four reports, published between 1998 and 2004, which provided the scientific basis EPA used to target its resources to address the highest priority PM research needs. These needs are being addressed by the Agency's particulate matter research program, with more than $500 million during the past 10 years committed by EPA in support of the highest priority research topics identified by the NAS. These funds have supported numerous research efforts by EPA's intramural laboratories, as well as extramural researchers funded through our competitively awarded Science to Achieve Results (STAR) program, our PM Research Centers, and interagency agreements with other federal agencies. EPA also coordinates closely with other federal agencies on PM research through the Committee on Environment and Natural Resources (CENR) Air Quality Research Subcommittee and its Interagency Working Group on Particulate Matter.

We learn about the potential health effects of PM through several different types of research, especially epidemiology and toxicology. Guided by the NAS and other advisors, the Agency has funded research in all of these areas. Epidemiologic studies supported by EPA and others provide key information in our evaluation of PM. This research includes population-based studies that evaluate potential associations between human exposure to PM and health outcomes, including death, hospitalization, illness, and potential precursors to illness. We have sponsored research on populations of tens to hundreds of thousands of individuals in the United States that evaluates the effects of long-term exposure to PM on illness and death. These include both cohort studies and panel studies. Other research uses a different design—called time-series studies—in which air pollution levels are tracked on a day-to-day basis and compared with daily variations in health statistics to evaluate the effects of short-term exposures to PM on health. These time-series studies included hundreds of communities and databases that describe millions of residents. Other epidemiologic studies attempt to identify factors affecting people's susceptibility and the role of co-pollutant exposures.

Toxicology studies, sponsored by EPA and others, provide both information to evaluate the strength and plausibility of the associations identified through epidemiology and hypotheses that form the basis of new epidemiological studies. Important

studies include those that evaluate the components of PM that may be producing toxicity, and the mechanisms by which such toxicity might occur.

These research efforts have resulted in literally thousands of published studies in the peer-reviewed literature over the past several years. In 2005, EPA prepared a report, Particulate Matter Research Program: Five Years of Progress, which highlighted the early results of EPA's substantial investment in PM. When it came time to prepare the science basis for the next evaluation of the PM standards (the 2004 Air Quality Criteria Document), more than 4000 articles from the peer-reviewed literature were reviewed—many of which came from research EPA had done in our laboratories or had funded through our STAR grants.

A second, and equally important, function of EPA efforts is the synthesis and integration of these thousands of individual "acts of science" to provide a clear characterization of our knowledge and the degree to which we still are uncertain about aspects of PM health and environmental effects. We have a scientifically rigorous process by which we evaluate and interpret this important body of knowledge and ensure that our interpretation of them is complete, transparent, unbiased, and consistent with the array of views in the scientific community. A fundamental step in the review of the National Ambient Air Quality Standards is the evaluation of scientific evidence and the preparation of scientific assessments, by the National Center for Environmental Assessment of the Office of Research and Development, known as "criteria documents." The development of criteria documents involves the review of thousands of peer-reviewed research publications, evaluation of those studies most relevant to the review of the air quality standards, and integration of the scientific evidence across disciplines. The body of evidence must be reviewed, evaluated, weighed and then accurately and objectively described to inform our decisions about National Ambient Air Quality Standards.

For the current PM review, EPA evaluated research studies that addressed a wide range of issues including PM toxicology, epidemiology, atmospheric chemistry, human exposure, and other areas such as environmental effects. Thousands of studies were reviewed and over 2000 studies were referenced in the criteria document, many of which were conducted or funded by EPA's Office of Research and Development. Considered together, these new studies significantly advanced our understanding of PM's potential effects on public health and welfare and reduced the uncertainty associated with some important aspects of the science. Drawing on the evaluation of studies reviewed in the PM criteria document about health effects and dose-response, as well as information about exposures to PM, EPA also completed a risk assessment to estimate the degree to which various approaches for revising the standards would potentially affect the public health risks posed by PM. Further, the Agency prepared a document known as a "staff paper" that utilized the evaluation and characterization of scientific evidence in the criteria document together with the results of the risk assessment to help inform the policy judgments required in making decisions on the NAAQS.

Extensive independent external peer review was conducted on the criteria document, risk assessment, and staff paper by the Clean Air Scientific Advisory Committee (CASAC). CASAC, statutorily-mandated under the Clean Air Act, is a group of independent scientific and technical experts appointed by the Administrator to review EPA's evaluation and use of scientific and technical information related to air quality and make recommendations as appropriate. CASAC is made up of nationally-recognized scientists from a variety of relevant disciplines. For PM, CASAC was extensively involved in reviewing and commenting on several drafts of the PM criteria document, staff paper, and risk assessment. Their efforts, and those by EPA staff to address CASAC's comments, resulted in a PM science assessment that provides comprehensive, relevant information suitable to serve as the scientific basis for Administrator Johnson's decisions on the PM NAAQS.

Let me briefly highlight some scientific information available on particulate matter. First, as a scientist, I know that all scientific research includes aspects of uncertainty. For example, we often do not understand the mechanisms by which pollutants such as particulate matter produce health effects in the population. We know our measurements of environmental conditions and biological response contain some uncertainty due both to our understanding and technological limits. To have uncertainty is normal. Uncertainty is a factor to be characterized and considered in the evaluation of studies and other data. We always consider the strengths and limitations of the available evidence when drawing conclusions about what that evidence means for decision making.

For example, we highlighted the uncertainty in the evidence linking chronic exposure to $PM_{2.5}$ with premature mortality in the 1997 review of the PM NAAQS. In the next few years, EPA responded by funding a major reanalysis by independent investigators of two important long-term studies that used data from a Harvard Six

Cities cohort and an American Cancer Society cohort. The quality of the data was evaluated, and an extensive series of sensitivity analyses were performed using various statistical models to test for the influence of many potential co-variables. The results duplicated the association between levels of chronic exposure to $PM_{2.5}$ and premature mortality. These analyses were important in reducing our uncertainty about the consideration of these data in the standard-setting process. In addition, the analyses identified other avenues of research. For example, one study indicated that the estimated effects of fine particles appeared to vary with education level.

In another example of our efforts to tackle uncertainty, EPA sponsored a number of multi-city epidemiologic studies designed to address the limitations inherent in single-city studies. Multi-city studies allow the assessment of risks of mortality or hospitalization across cities, thus reducing uncertainty regarding the effects of local features, such as differing mixes of pollutants and climates, on the interpretation of study findings. The results of these multi-city studies provide additional evidence that levels of exposure to $PM_{2.5}$ are likely to be linked with serious health effects.

Another major area of uncertainty remaining from the previous review was the lack of demonstrated biological mechanisms or pathways by which PM exposure could result in the effects observed in population-based studies. An important factor in evaluating the associations uncovered in epidemiologic investigations is biological plausibility, i.e., whether there is a coherent way in which the reported association could be expected to occur in the body. As noted in our 2005 report, EPA-funded research has provided crucial insights into numerous hypothesized mechanisms; including evidence that exposure to particles may contribute to atherosclerosis development and affect cardiac rhythm, thus linking the findings of mortality in the epidemiologic studies to plausible biological mechanisms of toxicity.

Looking across the large landscape of study findings, our assessment of the research results for particulate matter finds evidence of a coherence of health effects associated with $PM_{2.5}$ across many types of study designs, biological endpoints and time frames. The body of evidence—the thousands of studies from a wide variety of disciplines we have evaluated with the help of CASAC—demonstrates that $PM_{2.5}$ exposure is likely causally associated with outcomes such as cardiovascular and respiratory morbidity and premature mortality from both epidemiologic and toxicology studies. Toxicology studies help us understand the mechanisms that provide some evidence of biological plausibility in the observations from epidemiological studies. We recognize that uncertainty exists, but uncertainty is not a barrier to decision-making; rather it is critical information to be factored into informed decisions.

We also recognize that science is not static. New studies on PM are being published in the peer-reviewed literature all the time. As a continuation of the scientific review process, EPA recently conducted a survey of the evidence reported in the scientific literature since completion of the literature review reflected in the 2004 criteria document. This new survey includes some 700 additional studies and has emphasized the studies most relevant to the PM NAAQS decision. The provisional assessment of these new studies has only just been completed. To provide the public with an opportunity to review the survey results, we will provide notice of the completion of this survey and post the results on our Web sites. In brief, the provisional assessment concluded that taken in context, the new information and findings provide additional support regarding the health effects of PM exposure made in the 2004 PM Air Quality Criteria Document but do not materially change any of the broad scientific conclusions.

In summary, the Bush Administration is committed to the development and use of the highest quality science to inform environmental decision making. The mission of the Office of Research and Development is to develop, evaluate, and communicate relevant scientific information to the Administrator, and to assure that the Administrator is well informed of the nature, strengths, and limitations of this information. EPA has sponsored a targeted and effective research program on particulate matter and I am pleased to convey to you and others the value of this investment. We have made a great effort to evaluate and characterize the existing and new scientific results available on particulate matter, and I am personally pleased to share with you my views on this work. I look forward to addressing any questions you may have.

Thank you.

RESPONSES BY GEORGE GRAY TO AN ADDITIONAL QUESTION FROM SENATOR INHOFE

Question 1. The General Accountability Office found that EPA has fully implemented only 8 of the National Academy of Sciences' 34 recommendations in conducting its PM air quality review. One of its recommendations was to include a cal

culation of the total costs and benefits, including indirect benefits, in its regulatory impact analysis. It is well established that economic growth is associated with health, and conversely, economic costs and associated job losses is associated with increased mortality. In light of this, it is only possible to determine the net health benefit if these indirect costs are calculated. Does EPA intend to in the final RIA calculate the net benefits, including indirect disbenefits?

Response. EPA believes there are indirect effects that should be considered when the measured costs and benefits are sufficiently large. However, quantifying these issues is very difficult. Until better methods are developed to assess these second-order effects, EPA has focused on assessing the primary benefits and costs that result from a partial equilibrium analysis.

EPA uses a partial equilibrium analysis when assessing the benefits and costs of regulatory options. Partial equilibrium analysis, by definition, only looks at the direct effects of the regulation. It ignores the general equilibrium effects that may occur throughout the economy. Note that the partial equilibrium models ignore both the secondary benefits and the secondary costs that may occur. Partial equilibrium models are generally used when the costs and benefit impacts are small so that secondary impacts may be safely ignored.

However, if the regulation has sufficient impacts on benefits and costs, these secondary costs may be significant. There are at least two effects that must be measured to do this correctly:

The health-wealth tradeoff with costs: This literature suggests that costs of environmental regulation may be higher than the direct expenditures of a regulation. Specifically, raising costs also raises prices and reduces real wage income. This lower real wage reduces net income and may, therefore, reduce the overall health of workers. (The lower our income, the less health we can afford.) This is the effect mentioned in the question.

The health-wealth tradeoff with benefits: When our environment improves, we reduce the sick loss days of workers, and improve the overall health of the population. These changes increase labor productivity, raising the real wage. As the real wage increases, the populations' health increases. (As our income increases, we can afford to buy better health care, etc.)

In general, EPA assumes that these two effects roughly cancel. We have taken some exploratory looks at this issue and found this to be the case, though generalized conclusions cannot be made. These issues are also discussed in the context of EPA's analysis done under section 812 of the Clean Air Act amendments.

In addition to the above issues, it is our understanding that the NAS recommendation concerning indirect benefits focuses on whether any particular EPA analysis can and should try to capture potential indirect effects pertaining to changes in exposures. For example, the NAS gave as its first illustrative example of the indirect effects concept the possibility that air pollution regulations may change how fuels are made or how combustion devices are operated, which could in turn affect human health through other pathways.

RESPONSES BY GEORGE GRAY TO ADDITIONAL QUESTIONS FROM SENATOR VOINOVICH

Question 1. Dr. Gray, please put yourself in our position as the oversight Committee or the publics position whether a regulated industry, environmental group, or just a concerned citizen. Last week, we discussed at length the fact that EPA has put together an incomplete Regulatory Impact Analysis. Basically, we do not know the impact of revising the standards. Let me remind EPA that this information is required by Section 108 of the Clean Air Act: Administrator shall. . . issue to the States. . . information on air pollution control techniques, which information shall include data relating to the cost of installation and operation, energy requirements, emission reduction benefits, and environmental impact of the emission control technology.

We are told that a more complete analysis will be addressed in the final rule.

Today, we learn from GAO that EPA has implemented less than 25 percent of the National Academies recommendations. We are told more of these recommendations will be addressed in the final rule.

Chairman Inhofe has pushed EPA to consider the numerous new studies that have been completed since the criteria document was finished. We are told that this new science will be addressed in the final rule.

Don't we have an oversight responsibility and doesn't the public have a right to know what EPA is doing behind closed doors on this rule?

Response. EPA is committed to working with Members and committee staff to ensure the public is well-informed with regard to Agency rulemakings. EPA has been, and will continue to be, in regular contact with the committee regarding the $PM_{2.5}$ NAAQS and the associated RIA, and we believe our final analysis will be comprehensive, useful, and robust.

The Clean Air Act (Act) requires EPA to set a primary standard for each criteria pollutant that protects public health with an adequate margin of safety. As interpreted by the Agency and the courts, the Act requires EPA to base this decision on health considerations and forbids consideration of economic factors. Thus, while the final RIA can provide important insights into the costs and human health benefits associated with attaining a revised $PM_{2.5}$ NAAQS, EPA cannot use estimates of attainment cost to inform the NAAQS decision.

This prohibition against the consideration of cost in the setting of the primary air quality standard, however, does not mean that costs or other economic considerations are unimportant or should be ignored. The Agency believes that consideration of cost is an essential decision-making tool for the cost-effective implementation of these standards. Under the Clean Air Act, the impact of cost and efficiency are considered by the States during this process, when States are making decisions regarding what timelines, strategies, and policies make the most sense. The PM NAAQS final RIA is intended to inform the public about the potential costs and benefits that may result when any revised PM standards are implemented.

With regard to the new studies cited in your question, EPA's Office of Research and Development has conducted a survey and provisional assessment of relevant scientific information that became available since the completion of the 2004 Criteria Document, as discussed in EPA's notice of proposed rulemaking on the PM NAAQS. In summary, our provisional assessment concluded that the new studies do not materially change the broad conclusions of the 2004 document.

Question 2. Dr. Gray, Congress established the Clean Air Act to protect human health. Last week:

Georgia Commissioner Heiskell stated: As an elected official, I fear the lost tax revenues and increased stresses on local health services that lay-offs associated with our non-attainment status bring.

National Black Chamber of Commerce President Harry Alford stated: . . . the biggest health risk to African Americans anywhere. . . is poverty.

Now, I understand that EPA cannot consider costs. However, according to the GAO report:

Another recommendation (from NAS) that EPA. . . did not apply to the draft regulatory impact analysis concerns whether the proposed revisions to the particulate matter standards would have important indirect impacts on human health and the environment.

This is a health based standard and there are indirect, negative impacts on health from nonattainment designations that reduce jobs and increase energy prices. Why isn't EPA following the NAS recommendations and considering these impacts?

Response. Please see our answer to Question 1.

Question 3. Dr. Gray, does science dictate a particular number for the air quality standards?

Response. Uncertainty is inherent in science. As new data become available, our understanding of the health protectiveness of a given air quality standard grows, as does our understanding of the extent of uncertainties. That is why rarely, if ever, it is the case that a body of scientific evidence has the potential to dictate a particular number for an air quality standard. Science does provide the basic information used in setting air quality standards, and our analysis of the science includes consideration of the strengths and uncertainties in the available evidence. We know that our measurements of environmental conditions and biological responses contain some uncertainty due both to the limits of our understanding of the underlying process and available technology. EPA recognizes the importance of characterizing the implications of this scientific uncertainty.

In the review of the PM air quality standards, the scientific evidence has provided strong support that PM exposures can have adverse effects on public health and the environment. The science summarized in EPA's Criteria Document and used in EPA's risk assessment provided the scientific basis for the range of options recommended in EPA's staff paper. There is uncertainty even within this range of options. The strength of the scientific evidence as well as the uncertainties are necessarily considered by the EPA Administrator in proposing and promulgating final PM NAAQS.

Question 4. Dr. Gray, as I stated in my opening statement, I do not understand how EPA can revise the particulate matter standards when the public health benefit

is not fully understood. For example, there are areas of the country, namely Southern California, that will never be able to meet the Agency's proposal, not to mention even tighter standards. When EPA estimates health benefits, how do you take into account that this area will never attain the standards? Basically, does the Agency estimate real benefits?

Response. This Nation has an excellent record of rising to the challenge of reducing air pollution in a cost effective fashion. All areas have made significant progress in reducing air pollution even if some areas are still not yet in attainment of the current standards. The regulatory impact analysis that will accompany EPA's decision reflects the need to develop new ways and ideas for some areas to attain the proposed standard. To the extent that known controls are not sufficient to bring an area like Southern California into attainment by 2020, EPA has relied on information from a variety of sources on the effectiveness of new control strategies under development to project what the costs and benefits of attainment might be. While these estimates have additional uncertainty associated with them, they give the public an idea of the likely magnitude of the potential costs and benefits of attainment.

Because of its severe air pollution problems, Southern California has been one of the leaders in the development of creative ways to reduce air pollution by harnessing the free market. For example, the RECLAIM (Regional Clean Air Incentives Market) program has been in place since 1994 to harness market forces to reduce air pollution. See http://www.aqmd.gov/reclaim/reclaim.html for more information. Also, the California Air Resource Board (CARB) is now aggressively pursuing options for continuing to reduce air pollution. On April 20, 2006, CARB announced a new program for reducing air pollution from its ports which includes economic incentive elements. See http://www.arb.ca.gov/gmp/gmp.htm for more information.

Question 5. Dr. Gray, the GAO report states that the Agency has implemented less than 25 percent of the National Academies' recommendations for estimating health benefits. Does the Agency have a plan to implement all of the recommendations?

Response. EPA is committed to showing further progress in the final RIA, which will be published when the NAAQS is finalized this September. As noted in the GAO report "Particulate Matter: EPA Has Started to Address the National Academies' Recommendations on Estimating Health Benefits, But More Progress is Needed," EPA expressed to GAO that the Agency is committed "to further enhancing the transparency of the analysis by presenting clear and accurate references to the supporting technical documents, which detail the analytical assumptions and describe the data supporting the estimates [of our Regulatory Impact Analyses.]" GAO found that of the 34 detailed recommendations in the NAS report, the draft RIA implemented (in full or in part) 22 of them. Some of the remaining 12 recommendations will be addressed in the final RIA to accompany the final rule in September; others require further research or development. For example, over the past few years, EPA ran an expert elicitation designed to characterize uncertainty in the estimation of PM-related mortality resulting from both short-term and long-term exposure. This work has recently been completed and peer reviewed. The results from this expert elicitation are being applied to the benefits analysis in the RIA. However, it has not been possible to address all the NAS recommendations since the September 2002 publication of the report. In its report and its recommendations, the NAS recognized that additional research and resources were going to be needed to address some of its recommendations. And, in some cases, the recommendations were in fact conditioned on the availability of improved scientific data. EPA is committed to addressing these recommendations, including those on PM speciation, and improving its analyses as new research becomes available.

In its July 19, 2006, report, GAO acknowledged the progress EPA has made on implementing the recommendations while also noting that EPA needs to make more progress in addressing the National Academies' recommendations on estimating health benefits.

Question 6. Dr. Gray, how does EPA decide which studies to rely on and which ones to ignore? Do CASAC or EPA have any written criteria for determining which studies are more important than others? Any intention of developing this so that the NAAQS review process is more objective and transparent?

Response. We believe that the consideration of scientific evidence in the NAAQS review process is comprehensive, objective, and transparent. EPA conducts an extensive literature search to identify potential studies for inclusion in its science assessments. For example, EPA uses standard searches of systems such as MEDLINE in the National Library of Medicine, independent table of content searches by EPA staff, and input from the public and independent scientists outside EPA. These

search methods are summarized in the Criteria Document. The selection of studies for consideration in a NAAQS review, the strengths and uncertainties of individual studies, and the subsequent determination of studies to be given greatest weight are outlined and discussed in the science assessment. These selections and determinations are evaluated by the public and by CASAC at public meetings. The inputs from the public and CASAC are documented, considered, and incorporated as needed.

Question 7. What role did the White House and other Federal Agencies play in setting the NAAQS proposal?

Response. Under Executive Order No. 12866, "Regulatory Planning and Review," signed by President Clinton in September 1993, the Administrator of the Office of Information and Regulatory Affairs (OIRA) carries out a regulatory review process on behalf of the President. The objectives of this Executive order are to enhance planning and coordination with respect to both new and existing regulations; to reaffirm the primacy of Federal agencies in the regulatory decision-making process; to restore the integrity and legitimacy of regulatory review and oversight; and to make the process more accessible and open to the public. In general, involvement by other Federal agencies or the White House offices occurs during the routine interagency review process, when the Agency is developing its proposal and before making a final regulatory decision. Following this procedure, the proposal package for the PM NAAQS went through the interagency review process, which was managed by the OIRA and included input from various agencies and offices throughout the executive branch.

Once EPA has issued a proposal to the NAAQS, the Agency evaluates public comments received on the proposal and develops a draft final rule. The draft will then be subject to the interagency review process, per Executive Order 12866, which includes input from across the executive branch.

STATEMENT OF DANIEL GREENBAUM, PRESIDENT, HEALTH EFFECTS INSTITUTE

Mr. Chairman and members of the committee, thank you for the opportunity to testify before you today on the health effects of particulate matter (PM). I come before you as the President of the Health Effects Institute, a non-profit, independent research institute funded jointly and equally by the U.S. EPA and industry to provide high-quality, impartial science on the health effects of air pollution. For over 25 years we have conducted targeted research on a variety of pollutants and health effects, and I am pleased to summarize our understanding concerning PM and health for you today.

I also had the privilege to serve from 1998 until 2004, as a member of the National-Research Council Committee on Research Priorities for Airborne Particulate Matter, a congressionally chartered panel that both set priorities for national PM research, and monitored the progress in implementing those priorities by U.S. EPA and other public and private agencies.

I would like today to briefly highlight three topics of direct relevance to the current discussion of U.S. EPA's proposal for revisions to the National Ambient Air Quality Standards for PM (the "PMNAAQS"):

- Science progress we have made since 1997,
- The most recent findings on the relationships between different levels of ambient PM and health effects (so-called "concentration-response"), and
- Keyscience needs going forward.

PROGRESS SINCE 1997

Since Congress identified the need for substantial enhanced research on PM in the wake of the 1997 $PM_{2.5}$ NAAQS decision, established the NRC Committee, and appropriated substantial new funds for PM research, much progress has been made in answering key questions for the current NAAQS review process, and for future ones.

Specifically:

- We know much more about the sources and transport of fine particles, and about personal exposure to those particles, especially for sensitive groups like the elderly and children.
- We have conducted the first multi-city epidemiology studies of effects, and analyzed and reanalyzed many of the major studies of human effects, finding that in general the earlier studies were well done and could be confirmed. At the same time there has been some evidence that the population health effects we had seen in those earlier studies may in some cases be smaller than previously thought.

• Unlike in 1997, we now have numerous laboratory, animal, and human toxicology studies that have begun to indicate potential biological mechanisms by which PM may cause health effects, especially new findings of effects on the heart and circulatory system. Although we have made progress, most science observers would agree that there is still much to learn about the mechanisms by which PM may cause these effects.

Although there continue to be, as there always are, important questions about PM that need further research, I think Congress, the Federal Government, and the scientific community can take tremendous pride in the substantial progress that has been made.

The "Concentration-Response" Relationship: Ambient PM Levels and Health Effects

Among the most important questions addressed over the past few years is the question of whether exposure to PM has been shown to have health effects at.all levels of pollution—ie. down to zero—or whether there is a "threshold" below which no effects are expected. This question is, of course, central to deciding at what level to set a NAAQS. There are two major types of epidemiologic studies that have been done—of short term effects and long term effects—and I would like to briefly review what these studies have shown us.

Short Term Effects

In 1997, there were studies of daily changes in air pollution and health effects in a number of individual cities (so-called "daily time series studies"). Since then, scientists have conducted much more rigorous multi-city studies of daily air pollution and health, most notably the National Morbidity Mortality and Air Pollution Study (NMMAPS) funded by HEI and led by investigators at the Johns Hopkins Bloomberg School of Public Health. That study examined daily changes in air pollution and health in the 90 largest U.S. cities. To answer the question of whether there is a threshold for effects, the investigators analyzed mortality and pollution levels across the 20 largest cities and the 90 largest.

In brief, as shown in Figure 1, they found that there appeared to be a linear relationship between mortality and air pollution down to the lowest measured levels for all causes of mortality, and for deaths from heart and lung disease, without an apparent threshold. There did appear, however, to be a threshold for the effect of PM on "other" causes of mortality (e.g. non-respiratory cancer, liver disease). The HEI Review Committee, which intensively peer reviews all HEI research, advised "caution in drawing conclusions from the apparent absence of a threshold" for all-cause and cardiopulmonary mortality, for a number of statistical and analytic reasons. They noted however that "the reported associations are at ambient concentrations well below the current U.S. daily standard . . . thus the ambient concentration level at which any threshold might exist is likely to be correspondingly low."

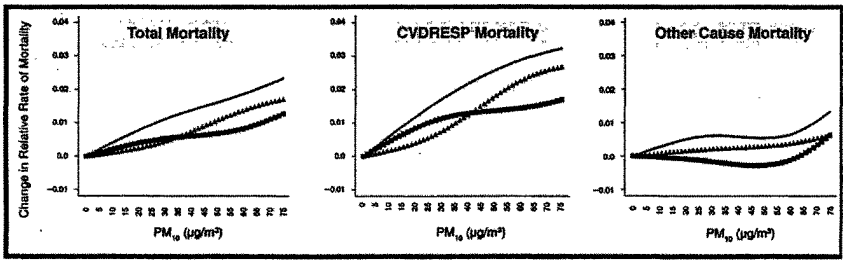


Figure 1. Short Term (Daily) Effects
National Morbidity, Mortality and Air Pollution Study (NMMAPS)
20 largest US cities (Daniels et al HEI 2004)

Long-Term Effects

In 1997, there were two principle studies of the effects of longer term exposure on mortality, the Harvard Six Cities Study and the American Cancer Society Study (ACS), which examined death rates among thousands of individuals living in cities with varying levels of pollution. Since that time, although there have been other long-term studies published there are still very few, and much attention has focused on HEI's Reanalysis of these two studies and on extended analyses in the American Cancer Society Study population (which still covers the broadest national popu-

lation). In both of these efforts, initial analyses have examined the "concentration-response" relationship between levels of pollution in each community and levels of health effects.

HEIReanalysis—At the request of Congress (in the FY 1998 Appropriations Bill), U.S. EPA, and others, HEI gained access to all underlying data in the two studies and selected an independent investigator—Dr. Daniel Krewski—and his team to conduct a detailed audit and reanalysis. Their work, which was also intensively peer reviewed by the HEI Review Committee, tested the original studies against a wide variety of alternative explanations about why people in the most polluted cities would have higher rates of premature mortality. In the end, the investigators and HEI's Review Committee agreed that these alternative analyses did not change the original findings of associations between PM and premature mortality, although there were new findings as well about an association of mortality with sulfur dioxide.

Dr. Krewski and his team also conducted an initial analysis of the "concentrationresponse" relationship between PM levels in each of the cities and relative risks of mortality. Figure 2 presents the results, summarizing for each community (signified by a point on the graph) the annual air pollution level and the risk of death due to heart and, lung disease. They then attempted to estimate the "average" relationship across all of the communities (the solid line) and the range of uncertainty around that average (the "95 percent confidence intervals" indicated by the dashed lines). As you can see there is some "scatter" in the data, especially at the highest and lowest PM levels studied, but also an overall trend of increasing mortality risk with increases in pollution levels starting at relatively low levels. In reviewing this initial analysis, the HEI Review Committee found that "for all-cause and cardiopulmonary mortality, the results show an increasing effect across the entire range of fine particles or sulfate but no clear evidence either for or against overall linearity."

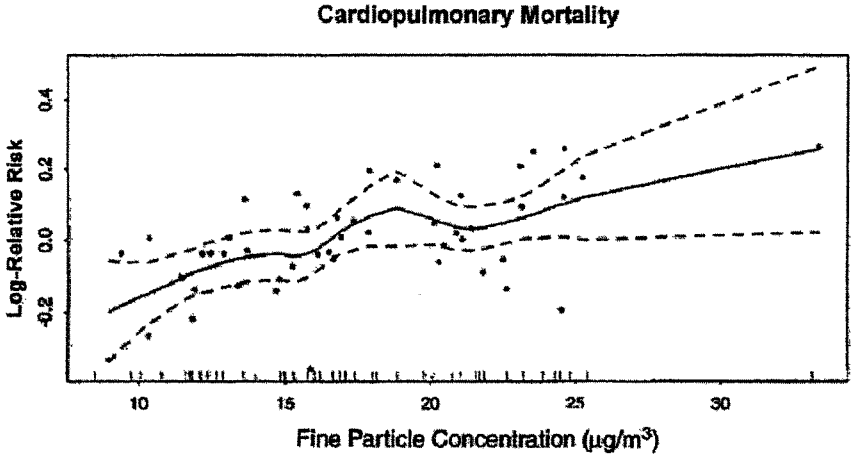


Figure 2 Long Term Effects
American Cancer Society Cohort
150,000 Individuals, 50 cities
HEI Reanalysis Results (Krewski, et al 2000)

Extended Analyses in the American Cancer society Cohort Following the reanalysis, the original investigators for the ACS study led a broad team of experts in an extended analysis of the data, including additional follow-up of more recent deaths among the study population, and using new $PM_{2.5}$ data from monitors installed since 1997. That study found results similar to those found in the Reanalysis and also conducted analyses of the "concentration-response" relationship (shown in Figure 3). This also shows a general, though less steep, upward trend in mortality with increasing pollution levels, with the largest uncertainty being found at the very lowest and very highest levels where there are fewer cities. The Investigators concluded that: "Within the range of pollution observed in this analysis, the concentration re-

sponse function appears to be monotonic and nearly linear. This does not preclude a leveling off (or even steepening) at much higher levels of air pollution."

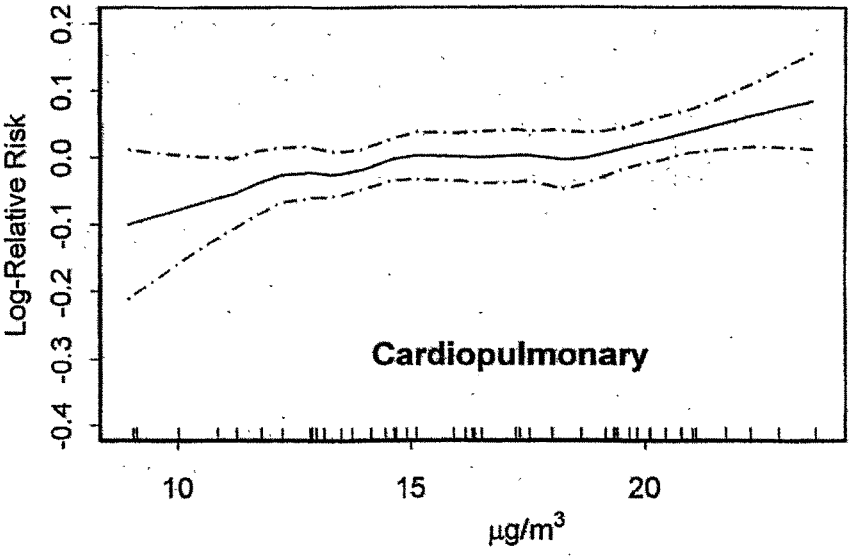


Figure 3. Long Term Effects
American Cancer Society Cohort
350,000 Individuals, 61 cities
Extended ACS Results (Pope, et al 2002)

Summary: PM-Mortality Concentration-Response

In sum, recent analyses of the relationship between ambient levels of pollution and mortality have found a generally increasing trend of mortality with increases in pollution across a wide range of locations. The strongest evidence that there is not a threshold for these effects comes from studies of short-term effects, where any threshold is likely to be well below the current ambient standards. The initial analyses of these relationships in long term studies also shows this general pattern, albeit with somewhat greater uncertainty at the lowest and highest levels.

KEY RESEARCH NEEDS LOOKING FORWARD

While we have made much progress in understanding PM exposure and health effects over the past decade, there continue to be, as there always are in science, important questions to be answered to help inform future decisions about ambient air quality standards and protecting public health. Two key areas needing continued attention are:

Continuous Improvement in the Statistics Used in Epidemiology

The analysis and reanalysis of studies on population health, air pollution and weather over the last decade have enhanced our ability to determine whether health effects can be tied to certain air pollutants. However, those same analyses have shown that the results can be significantly affected by the choices of statistical techniques and the assumptions made in each analysis. Looking forward, we need to pay continued attention to understanding the sensitivity of the results to different assumptions, quantifying the uncertainty of the results, and communicating clearly for each analysis both the results and the continuing uncertainties around those results.

Systematic Analyses of which PM Components and Sources May Contribute the Most to Toxicity

Perhaps no other question will need as much attention, and will have as much implication for future regulations, than determining whether some components of the complex mixture of PM are more toxic than other components. Ultimately, this data will be essential to ensuring that regulations and control strategies are targeted at reducing those emissions which will have the most public health benefit at the least cost. This has also become important in light of the current proposal for a PMNAAQS for "coarse particles" which has proposed to exclude certain particles from consideration even before the needed studies are complete.

To date, there have been some individual city analyses of toxicity of different components supported by U.S. EPA and others; but no systematic national effort to compare results from across the country and from epidemiology and toxicology studies. To fill that gap in time to inform a next round of PMNAAQS review, HEI has launched, with support from EPA and multiple industries, a sysatematic, multi-disciplinary effort which will:

- Conduct comprehensive, multi-city analyses of PM components and health
- Combine and compare epidemiology and toxicology across the country, and
- Provide the first-ever analyses of long-term effects of different PM components (all studies to date have looked only at daily changes in air pollution and health)

As indicated in both the NRC review of priorities for future PM and health research (NRC 2004), and in today's report of the Government Accountability Office concerning data needed for future PM benefits analysis, these studies will be central to ensuring that future PM actions are the most effective possible.

SUMMARY: PROGRESS MADE AND MORE TO BE DONE

In sum, we have made much progress since 1997 in answering key questions about whether and how PM can affect public health. Initial analyses have also helped us better understand the "concentration-response" relationship between levels of ambient air pollution and health effects and the generally increasing effects with increasing levels of pollution. At the same, looking forward there continue to be important issues to be addressed to inform future NAAQS and regulatory decisions, especially around the toxicity of different component and sources of PM.

Thank you for this opportunity to testify; I would be pleased to answer any questions you might have.

RESPONSES BY DANIEL GREENBAUM TO ADDITIONAL QUESTIONS FROM SENATOR VOINOVICH

Question 1. Mr. Greenbaum, as you know, there is an ongoing debate about whether EPA must lower the annual standard. Your opinion is invaluable in this debate as an entity that sits squarely in the middle -funded by both industry and EPA. Clearly, the statute says that standards shall be set ″ in the judgment of the Administrator.″ In your opinion, is it reasonable from a scientific and health perspective for the Administrator to retain the annual standard at 15 and not lower it, does science dictate a particular number for the air quality standards?

Response. This is of course one of the major questions facing the Administrator as he makes final decisions on the NAAQS. As I noted in my testimony, the Health Effects Institute has not, since its inception over 25 years ago, taken a position advocating either changing or retaining a specific level of a standard. We do this so that no one can ever question our science as having been created solely for the purpose of supporting an advocacy position. We do, however, attempt to provide the best possible impartial interpretation of the science to help inform such decisions. In that spirit, I would like to re-iterate and strengthen several points I made on this question in the hearing:

(a) First, the science on the relation between different levels of air pollution—and whether there are health effects at all levels or a ″threshold″ level below which there are no effects—has improved since 1997.

(b) Second, as I noted in my testimony, these so-called ″concentration-response″ relationships have shown for short-term studies (that inform the 24-hour standard) a fairly consistent relationship between levels of air pollution and increases in premature mortality at levels below the current standard.

(c) Third, for longer-term studies such as the American Cancer Society Study (that inform decisions on the annual standard), there is also evidence of a relationship between air pollution and premature mortality that extends below 15, but the level

of uncertainty on that relationship is somewhat greater than for the short term studies.

(d) Finally, there is no widely accepted scientific method for setting the level of a standard. Rather, as the Clean Air Act envisions, decisions on levels of the standard are a public health policy judgment by the Administrator, in which he has to weigh evidence that suggests that there are effects below the current standard against the level of certainty or uncertainty surrounding that evidence (i.e. the degree to which he can be certain from the science that a lower standard would result in greater public health benefits than the current standard.)

Question 2. Mr. Greenbaum, please explain what you mean by this statement in your testimony: "there has been some evidence that the population health effects we had seen in those earlier studies may in some cases be smaller than previously thought." Since 1997 there have been extended reanalyses of a number of daily and long term studies of relationships between air pollution and health.

In the case of short term (daily) studies, after HEI investigators at Johns Hopkins discovered an issue with the software to conduct these studies in 2002, those investigators and many others revised their analyses using better techniques, and HEI was asked to intensively peer review those results and draw conclusions on the new findings. In that report HEl's Review Committee found, among other things, that for the major multi-city daily time series studies done in the United States and Europe—some of the most systematic and rigorous of these studies—the estimates of risk went down between 30 percent and 50 percent (although they were still statistically significant). For example, for the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) conducted by HEI investigators, the estimates of effects went from 0.4 percent increase in premature mortality per 10 u/m^3 of paniculate matter to 0.2 percent per 10 u/m^3. (Health Effects Institute. 2003. Revised Analyses of Time-Series Studies of Air Pollution and Health. Special Report. Health Effects Institute, Boston MA.) In the case of long-term studies (of people living for many years in more-and less-polluted cities), the effects have been smaller in some analyses and larger in others. HEI's reanalysis of the American Cancer Society (ACS), which audited and in general validated the results of the original study, found lower estimates of health risk when one includes other pollutants in the analysis. However, some recent analyses of the ACS data by HEI investigators using improved estimates of personal exposure have actually found higher estimates of effect (see: Jerrett, M; Burnett, R. T.; Ma, R.; Pope, C. A., Ill; Krewski, D.; Newbold, K. B.; Thurston, G.; Shi, Y.; Finkelstein, N.; Calle, E. E.; Thun, M. J. (2005) Spatial analysis of air pollution and mortality in Los Angeles. Epidemiology 16: 727-736.)

Question 3. Mr. Greenbaum, please comment on the peculiarity that no effect is found for people with higher education?

Response. As I mentioned in response to a similar question in the hearing, a careful reading of the full results of recent studies does not necessarily suggest that the studies have found "no effect" for people with a higher education. The HEI Reanalysis of the Harvard Six Cities and American Cancer Society studies was the first analysis to attempt to look at whether there were differential effects in people with different levels of education (which is generally an indicator of different levels of socioeconomic status). That analysis, and a subsequent extended analysis in the ACS study, found a distinctly higher effect on mortality for those with lower education (and likely lower socioeconomic status). The HEI Reanalysis also found, for those with more than a high school education, a positive but not statistically significant association with all- cause mortality (a relatively crude measure of mortality since it includes causes of death which we expect could not be related to air pollution). When one looks at more specific causes of death, one finds that even for those with higher education, there is an association of mortality for cardiopulmonary deaths, and especially cardiovascular deaths. (See Table 52 in Health Effects Institute. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Paniculate Air Pollution and Mortality: A Special Report of the Institute's Particle Epidemiology Reanalysis Project.) There are several reasons why these differences in effects among people of different levels of socioeconomic status might be seen. First, it is plausible that people of lower socioeconomic status have higher exposures to air pollution due to: living in more heavily polluted areas; the likelihood that they have less access to air conditioning; and the jobs they have which may involve more outdoor exposure. Second, it is also well known that poorer people have worse levels of nutrition and poorer access to quality medical care, which could reduce their underlying health and make them more susceptible to the effects of air pollution. Third, it is also possible that, although these studies have made extensive efforts to separate the effects of "being poor" from the effects of "being exposed to

air pollution", there are some results in these studies that continue to reflect the impacts of poverty on health.

STATEMENT OF ROGER MCCLELLAN, ADVISOR, TOXICOLOGY AND HUMAN HEALTH RISK ANALYSIS

EXECUTIVE SUMMARY

- The scientific basis for policy decisions on setting the $PM_{2.5}$ National Ambient Air Quality Standard remain highly uncertain.
- The continued use of the $PM_{2.5}$ indicator is a default decision driven by EPA's past emphasis on regulatory compliance monitoring—"monitor that which is regulated." As a result, there is no database for considering alternative PM indicators that might target specific PM constituents or exclude certain constituents.
- The scientific database provides a basis for the Administrator making policy choices for a $PM_{2.5}$ NAAQS with 24-hour averaging time concentration in the range of 25 to 35 m^3, with a 98th percentile form, and an annual standard in the range of 12 to 15 m^3.
- The scientific database for policy decisions on setting a $PM_{10\text{-}2.5}$ NAAQS is very weak and highly uncertain. A science-based decision, as contrasted with a judicial decision, would be to continue with a PM_{10} NAAQS.
- There are major uncertainties in risks associated with exposure to ambient $PM_{2.5}$ at current levels and the benefits of reducing $PM_{2.5}$. These uncertainties need to be clearly documented and conveyed in numerical calculations used for policy decisions and in the Agency's final Regulatory Impact Analysis.
- Expert elicitations of opinions on $PM_{2.5}$ risks are very likely flawed with a blurring of the distinction between scientific evaluation and policy choices. Scientists, as do all citizens, have values that influence choices of standard setting options. However, scientific evaluations should be as free as possible of concern for the ultimate policy decisions.

Good Morning, Mr. Chairman and members of the subcommittee. Thank you for the invitation to present my views on the U.S. Environmental Protection Agency's current review on the National Ambient Air Quality Standards (NAAQS) for Particulate Matter (PM).

MY BACKGROUND

My biography is attached to this statement. Since 1999, I have served as an Advisor to public and private organizations on issues related to air quality in the ambient environment and workplace drawing on more than 45 years of experience in comparative medicine, toxicology, aerosol science, and risk analysis. I served as President of the Chemical Industry Institute of Toxicology in Research Triangle Park, NC from 1988 to 1999, providing leadership for a research program directed to understanding the mechanisms of action of chemicals in producing either beneficial or harmful effects on humans. I was with the Lovelace organization in Albuquerque, NM from 1966 to 1988, providing leadership for one of the World's major research programs directed toward understanding the potential human health effects of inhaled materials.

The testimony I offer today draws on my experience serving on numerous scientific advisory committees. This has included service on many EPA advisory committees from the origin of the Agency to date, including the Clean Air Scientific Advisory Committee (CASAC), which I chaired from 1988 to 1992, all of the CASAC PM Panels as well as CASAC Panels that considered other criteria pollutants. My involvement in advising EPA on the setting of NAAQS for criteria pollutants began with my chairing in 1977 and 1978 an ad hoc committee to review the first lead criteria document, a committee that was required since the Congress had not yet authorized creation of CASAC. I also served on the National Academy of Sciences/National Research Council (NAS/NRC) on Research Priorities for Airborne Particulate Matter and the earlier NAS/NRC Committee that produced the report "Science and Judgment in Risk Assessment." It is important to note that the testimony I offer today reflects my own views and is not being offered on behalf of any of the Committees I have served on for the EPA, the NAS/NRC nor for any other agencies or firms.

SETTING NATIONAL AMBIENT QUALITY STANDARDS

Each NAAQS consists of four elements: (a) an indicator (such as $PM_{2.5}$), (b) an averaging time (such as 24 hours or annual), (c) a numerical level (such as 65 m^3

for $PM_{2.5}$ averaged over 24 hours), and (d) a statistical form (such as a 98th percentile). The indicators for five of the criteria pollutants are for measurement of the mass concentration of specific chemicals such as O_3, SO_2, NO_2, CO and Lead. Only in the case of particulate matter is the indicator based on the mass concentration of airborne particulate matter in a specific size range, irrespective of the chemical composition of the PM.

Under the Clean Air Act, the EPA Administrator is required to review the NAAQS for the criteria pollutants at 5-year intervals to evaluate whether or not the four elements of the NAAQS are still deemed to be acceptable based on current scientific knowledge as it applies to the assessment of public health risks. In practice the interval between reviews has been longer. The process for review and promulgation of a NAAQS, either continuation of the existing standard or establishing a new NAAQS, consists of multiple phases. The initial phase, which is obviously on-going, consists of conduct of research on the various criteria pollutants. This includes a broad spectrum of activities; understanding emissions of pollutants, transport and transformation of pollutants in the atmosphere, ambient measurements of pollutants, estimation of personal exposures to pollutants, assessment of toxic effects and mechanisms of action in cells, tissues and animals, conduct of controlled exposure studies to pollutants in human volunteers and epidemiological investigations of human populations. Most of the research is funded by the EPA, some in the Agency's own laboratories and some in academic and other laboratories, the National Institutes of Health and, to a modest extent, private industry. The dominance of Federal Government support of research on criteria pollutants relates to their effects being of broad societal concerns with the pollutants, by and large, having no unique industrial emission source.

The findings of this research are used by the EPA's Office of Research and Development to prepare a criteria document (CD). Each CD traditionally has been essentially an encyclopedia of everything known about a given criteria pollutant and is used as a basis of information for the preparation of a Staff Paper (SP) by the EPA's Office of Air Quality Planning and Standards. This is a Policy Assessment of Scientific and Technical Information; in short, an integration and synthesis of the information in the CD that is most relevant to setting the four elements of a NAAQS. In recent years, the Staff Papers have made substantial use of risk assessments for the criteria pollutant being considered. These risk assessments have been conducted by a single EPA Contractor organization. The various versions of the CD and SP are released to the public with an invitation to provide comments as a basis for improving the documents.

Throughout this process, a Clean Air Scientific Advisory Committee Panel, operating as an element of the EPA's Science Advisory Board, is involved in reviewing and advising on the scientific content of both the CD and the SP, including the related risk assessment. This has typically involved several revisions. Prior to the current cycle of PM review, the CASAC Panel sent a closure letter to the EPA Administrator when the CASAC was of the opinion that the revised documents were suitable for use by the Administrator in promulgating a NAAQS. In the current review, the "closure letter" process was abandoned.

At the next step, the Administrator proposes, via a Federal Register Notice, a NAAQS including specific proposals for each of the four elements of the NAAQS; the indicator, averaging times, numerical levels and statistical forms. Comments are solicited from the Public with the opportunity to submit written comments to a specific Docket. In the current PM review, the CASAC PM Panel offered written comments on the Administrator's proposal.

The next step is for the Administrator to promulgate a NAAQS consisting of the four elements discussed previously. I purposefully do not use the phrase "final step," because the Courts may have a role in deciding whether the Administrator's proposed NAAQS will stand. The NAAQS are to be based on the available scientific information reviewed in the CD and SP and summarized in the notice of proposed standards. The primary, health-based NAAQS are to be set at a level that will protect public health, including sensitive populations, with an adequate margin of safety. The Administrator is precluded from considering cost in the setting of the NAAQS.

At this point, I would like to emphasize that there exists no absolute and unambiguous scientific methodology that can determine which specific indicator, the precise averaging time, numerical level or statistical form that will be adequate to protect public health. The available scientific information can inform the NAAQS decisions, however, the Administrator must ultimately use policy judgment in making decisions on each of the four elements from among an array of scientifically acceptable options including consideration of their attendant scientific uncertainties.

Once the NAAQS are finalized, individual states have responsibility for planning and taking actions to meet the NAAQS. This includes the formal step of preparing "State Implementation Plans (SIPs). In developing strategies for meeting the NAAQS, the States can give consideration to costs in setting the pace for achieving the NAAQS. However, attainment of the NAAQS cannot be postponed indefinitely.

EPA ADMINISTRATOR MADE POLICY CHOICES CONSISTENT WITH THE SCIENCE

At this juncture, I note that I personally find acceptable the Administrator's policy choices for the PM NAAQS, as published in the Federal Register (January 17, 2006) from among an array of science-based options, to be acceptable. Specifically, I find scientifically acceptable his proposal to use (a) a $PM_{2.5}$ indicator with a 24-hour averaging time and a reduction in the concentration level from 65 m^3 to 35 m^3 with a 98th percentile form, (b) retention of the $PM_{2.5}$ annual standard at 15 m^3 with additional constraints on the use of spatial averaging, and (c) use of a $PM_{10\text{-}2.5}$ indicator with a 24-hour averaging time concentration level set at 70 m^3 with a 98th percentile form. I support the exclusion of any ambient mix of $PM_{10\text{-}2.5}$ where the majority of coarse particles are rural windblown dust and soils and PM generated by agricultural and mining sources.

Of these several policy choices, I have the greatest reservation concerning the proposal for a $PM_{10\text{-}2.5}$ indicator with a 24-hour averaging time concentration level set a 70 m^3 with a 98th percentile form. The scientific basis for the proposed $PM_{10\text{-}2.5}$ standard is very weak and uncertain. I would have personally preferred to see the PM_{10} standard continued to provide public health protection from particulate matter mass in the $PM_{10\text{-}2.5}$ range. However, EPA personnel have related that this option has been precluded by Court decisions.

SELECTION OF A PM INDICATOR—CHAINED TO THE REGULATORY COMPLIANCE MONITORING LAMP POST

The primary scientific data used to select indicators for PM NAAQS has been derived from epidemiological investigations. Prior to 1970, there was limited regulation of particulate matter in air pollution. Limited monitoring, relative to that being carried out today, was conducted using relative crude metrics of Black Smoke and Total Suspended Particulates (TSP). TSP was the mass of particulate matter, not identified as to chemical form, collected on a filter in a high volume air sampler. This included material up to about 40 m in size. Scientists studied the relationship between the air concentration of these TSP measurements and increases in health effects. This epidemiological data provided the basis for setting the 1971 PM NAAQS with TSP as an indicator. The 24-hour averaging time standard set at 260 m^3, not to be exceeded more than once a year, and an annual standard set at 75 m^3, annual geometric mean. The TSP indicator then became the "law of the land" and TSP began to be routinely monitored to determine regulatory compliance.

During the 1970s and early 1980s, an increased awareness emerged on the role of particle size in determining the fraction of inhaled particles that would be deposited and where they would be deposited in the respiratory tract. This led to some groups making measurements of ambient air particulate matter mass in different size fractions; less than 15 m, less than 10 m, less than 2.5 m and less than 1 m. However, the primary epidemiological data in the 1980s that could be used for standard setting was TSP—remember TSP was required to be measured for regulatory compliance.

In 1987, the PM NAAQS indicator was changed from TSP to PM_{10}. The choice of PM_{10} was heavily influenced by a decision in the international community to use a PM_{10} metric rather than a PM_{15} metric. The United States followed suit. Much of the epidemiological evidence for setting a PM_{10} NAAQS was based on extrapolations from epidemiological studies using the TSP monitoring data. The PM_{10} primary standards were set at 50 m^3, expected annual arithmetic mean over 3 years, and 150 m^3, 24-hour average, with no more than one expected exceedance per year. With the promulgation of the PM_{10} indicator the regulatory compliance monitoring shifted from TSP to PM_{10}. Unfortunately, ambient air monitoring of PM_{15}, $PM_{2.5}$ and PM_{10} was essentially discontinued. Obviously, it would have been expensive to continue, and, after all, it was not required for regulatory compliance.

In the early 1990s, epidemiological data began to be published on the association between elevated $PM_{2.5}$ levels and their association with increased health effects. The data came principally from the Harvard Six Cities study that fortunately had included in its early years measurements of PM_{10} and $PM_{2.5}$. Other analyses were published based on an American Cancer Society cohort taking advantage of fragmentary $PM_{2.5}$ ambient monitoring data. Other investigations conducted using the PM_{10} ambient monitoring data were extrapolated to a $PM_{2.5}$ indicator. These data

provided the basis for promulgating a $PM_{2.5}$ NAAQS in 1997. The $PM_{2.5}$ NAAQS were set at 15 m^3, annual arithmetic mean, and 65 m^3, 24-hour averaging time with a 98th percentile of concentration at each population-oriented monitor. Associated with this was a change in the regulatory compliance monitoring network to emphasize $PM_{2.5}$ mass measurements without regard to chemical composition. Because a PM_{10} mass NAAQS was still in place measurements of PM_{10} mass, not characterized as to chemical composition, continued. Using the difference between the PM_{10} mass measurements and $PM_{2.5}$ mass measurements, it was possible to estimate $PM_{10\text{-}2.5}$ mass concentrations.

At various times there has been an interest in measuring PM sulfate mass concentration, a secondary pollutant arising in the atmosphere from conversion of SO_2 gas. There have also been some short-term monitoring campaigns in which extensive chemical characterization of a number of particulate matter constituents have been measured. However, the extent of this monitoring data is limited in comparison with that developed for regulatory compliance purposes on PM_{10} mass and $PM_{2.5}$ mass, not characterized as to chemical composition. Indeed, to date the database on specific PM constituents has been insufficient to set a NAAQS for a specific PM component. Obviously, Lead is an exception. Likewise, the data on specific PM constituents were not viewed as to exclude any constituent from regulation.

The most recent CD and SP focuses on the $PM_{2.5}$ indicator. The focus on $PM_{2.5}$ was not based on any careful scientific analysis that led to the conclusion that $PM_{2.5}$ mass, not identified as to chemical composition, as the most appropriate metric to relate to an increase in health effects. The simple fact is that because of the EPA's emphasis on regulatory compliance monitoring, the only PM air quality metrics that could be evaluated epidemiologically were PM_{10} mass, $PM_{2.5}$ mass and to a lesser extent PM sulfate and to an even lesser extent, $PM_{10\text{-}2.5}$. I will be so bold as to say the focus on $PM_{2.5}$ mass, irrespective of chemical composition, was a default decision, not a science-based decision.

My discussion so far has focused on epidemiological evidence without considering the results of toxicological studies using cells, tissues or laboratory animals. As a toxicologist, I wish I could give more emphasis to the conduct and interpretation of toxicological studies. However, such studies have a very limited role in the PM NAAQS setting process. Although such studies can use new tools of modern molecular and cellular biology and genomics, the results are not necessarily relevant to setting the NAAQS. The challenges of extrapolating from laboratory animals to humans, from high to low levels of exposure, from studies of a few days or even a few months to human lifetimes and from studies of a few normal healthy young animals to large human populations including individuals with cardiopulmonary disease, principally from smoking, are substantial. At best, the toxicological investigations can help provide some guidance to the design and conduct of epidemiological investigations. The toxicological methods are simply to blunt and yield results that at best can be extrapolated qualitatively to human populations. I know of no scientific methods for using the results of toxicological studies with PM, not characterized as to chemical composition, or those conducted with specific PM constituents to develop quantitative numerical standards that are at the core of PM NAAQS.

A SHIFT IN MONITORING STRATEGY TO FACILITATE EPIDEMIOLOGICAL EVALUATIONS

What are the prospects for the next PM NAAQS review in 5 years including a rigorous evaluation of specific PM constituents? Without a major revolutionary change in the EPA's approach to ambient air monitoring, I think it will be more of the same. In short, because of the past focus on measuring $PM_{2.5}$ and $PM_{10\text{-}2.5}$, these metrics will continue to be evaluated in future epidemiological studies. Because of the substantial and continuing improvements in air quality, including $PM_{2.5}$, PM_{10} and $PM_{10\text{-}2.5}$, it will be even more difficult to detect associations between these PM mass metrics and health effects. Future epidemiological studies will also be challenged due to continuing reductions in cardiopulmonary disease related to reductions in the primary risk factor for these diseases—Cigarette Smoking.

How can the prospects for improved epidemiological investigations be changed? If the EPA, in cooperation with States and Municipalities, radically modifies its ambient air monitoring network over the next 2 years, it may be possible to have the results of improved epidemiological studies in 8 to 10 years. The development of an improved ambient air monitoring network will require some tough decisions. It is obvious that the expense of an altered monitoring network will require that only a modest number of PM constituents be measured in multiple cities in different regions across the United States. Some clear candidates would be sulfates, nitrates, organic carbon, elemental carbon, silica and some specific metals for which concern may exist as to their potential hazard. It is essential that all of the criteria pollut-

ant gases, ozone, SO_2, NO_2 and CO, continue to be measured. With a richer array of monitoring data available it may be possible to test hypotheses as to the relative potency of the various PM constituents as well as the gaseous pollutants. In any long-term studies, it will be crucial to have accurate smoking history data if the very small potential effects of air pollution are to be separated from the large cardiopulmonary impacts of cigarette smoking. In addition, because of the relationship between PM-associated hydrocarbons and volatile and semi-volatile hydrocarbons these should be measured. In my opinion, it will be futile to measure dozens of individual chemical species with the view that these measurements could be useful in future epidemiological studies. The current highly uncertain signal of air pollution associated health effects is so small that "teasing out" effects related to any single PM chemical constituent will be extraordinarily challenging.

SELECTION OF AVERAGING TIMES, NUMERICAL LEVELS AND STATISTICAL FORMS

Having selected an indicator, it is necessary to proceed to decisions on the averaging times, numerical levels and statistical forms for the NAAQS. These three elements are inter-related and are set based on the epidemiological database. The averaging times are driven by the temporal characteristics of the monitoring data, 24-hour measurements that can be aggregated to yield annual values which, in turn, are used in the epidemiological investigations. Hence, it is reasonable to use 24-hour and annual averaging times.

The selection of specific numerical levels for the 24-hour standard has been guided primarily by considering the results of epidemiological studies of the association between daily changes in the PM indicator and changes in mortality (all cause, cardiovascular and respiratory mortality). The power of these studies is directly related to the size of the population being studied and the number of days being monitored. Thus, results can only be developed for quite large cities. This approach would not be feasible for small communities and rural areas.

The primary input for establishing the $PM_{2.5}$ annual standard comes from long-term follow-up of cohort populations, the Harvard Six City Study of about 8,000 individuals initiated in 1979 and the American Cancer Society cohort assembled starting in 1979. In these studies, sophisticated statistical techniques have been used to attempt to tease out an association between differences in $PM_{2.5}$ ambient concentrations in different communities and the risk of death from various diseases. The analyses are very complicated because of the numerous factors that can influence the death rate including age, cigarette smoking, work history, education, socio-economic status, exposure to other pollutants as well as other factors.

The results of the cohort epidemiological studies are typically reported as a linear coefficient of increase in relative risk per 10 m^3 of $PM_{2.5}$ using whatever $PM_{2.5}$ monitoring data are available for the specific cohort. Thus, for the studies initiated in 1979, this may be $PM_{2.5}$ measurements made in 1979-1983. Recall that in the 1980s, there was a move to regulate PM_{10} measurements of $PM_{2.5}$ were discontinued and not re-instituted until after the $PM_{2.5}$ NAAQS was promulgated in 1997. The $PM_{2.5}$ exposure of individuals in the cohort prior to 1979 is unknown although it is well recognized that in most areas air quality has substantially improved since 1970.

A major challenge in analyzing and interpreting the results of the cohort studies relates to the uncertain role of pollution exposures for the individual populations prior to initiation of the studies and the uncertainty in the statistical models used to attribute relative risk to the various risk factors including $PM_{2.5}$. The small size of the $PM_{2.5}$ relative risk poses a special challenge. This includes the difficulty of determining the shape of the exposure-health response relationship extending from past high levels down to current levels. Especially vexing is the issue of whether a threshold does or does not exist in the exposure-health response relationship. In my view, the exposure-response relationships are highly uncertain in the range of typical ambient $PM_{2.5}$ concentrations in the United States. The substantial uncertainty in the applicability of the $PM_{2.5}$ exposure-health response coefficients at current ambient concentrations requires caution in calculating either $PM_{2.5}$ associated risks or the benefits of any reductions in $PM_{2.5}$ concentrations.

EXPERT ADVICE ELICITATION

In an attempt to better characterize the uncertainties in $PM_{2.5}$-associated health risks and, conversely, the benefits in reductions in $PM_{2.5}$, some individuals have suggested the use of an "expert advice elicitation" approach. I am familiar with this approach having served as one of the five experts in EPA's pilot project to elicit opinions on the relationship between $PM_{2.5}$ exposure and death. I have also participated in such approaches in the initial stages of planning and interpreting safety assessment studies. I think the expert opinion elicitation process may have merit

in obtaining a qualitative assessment of the impact of exposure to hazardous materials. However, I have serious reservations as to its use in eliciting quantitative characterizations of risk for various levels of $PM_{2.5}$ exposure for different populations in different parts of the United States.

The interviewers eliciting the expert opinions play a major role in determining the outcome of the process. In the session I participated in, I found the interviewer focusing on eliciting quantitative linear exposure-response coefficients. Since it is my professional opinion that it is very unlikely that a linear relationship exists between $PM_{2.5}$ exposure and health responses down to and including current ambient levels, the interview and the follow-up discussions proved frustrating for both me and the interviewer. In short, the sponsor (in this case, the EPA) can influence the interviewer to frame a series of questions that will yield a pre-determined answer. In my case, I felt the desired answer was what linear risk coefficient (exposure health-response) would I prefer.

I am also concerned about the process used to select experts for participation. In my opinion, the process should be very transparent with regard to the criteria used to include or exclude individual experts from a Panel. My concerns extend to the inclusion of individuals who may have conducted and reported on the key studies being used in the expert opinion elicitation process. It is human nature to want to have one's own data and analyses used in the same manner as originally reported.

Any additional concern with the process is the approach of using secondary interviews in an attempt to gain consensus from the experts as to the outcome. I understand that was done with the full-scale expert elicitation panel whose input is to be used in the final regulatory impact analysis. A major challenge in any elicitation of expert advice is separating the individuals science-based input from their personal sources with regard to a policy outcome. In my opinion, the results of that expert elicitation are likely to be seriously flawed. I would urge the Administrator to not use the results of the expert opinion elicitation as input for quantitative estimates of risks/benefits associated with $PM_{2.5}$ exposure. Such an approach is not a substitute for more rigorous uncertainty analysis that attempts to characterize all the factors that impact on estimating risks of $PM_{2.5}$ exposure and the benefits of reductions in $PM_{2.5}$ exposure.

As an alternative to expert opinion elicitation, I urge CASAC to document the scientific views of each of the CASAC PM Panel members with regard to quantitative aspects of the PM NAAQS. This approach was used in the previous PM review that concluded with promulgation of the 1997 $PM_{2.5}$ NAAQS. A copy of the table included in the CASAC PM Panel's "closure letter" is attached. As may be noted, individual Panel members had a wide range of views with regard to setting the $PM_{2.5}$ NAAQS. I would personally prefer to see each of my scientific colleagues express their individual science-based opinions rather than have CASAC Panel participants cajoled to reach a consensus.

SCIENTIFIC EVALUATIONS VERSUS POLICY DECISIONS

A major challenge I see for all scientists, and especially for CASAC PM Panel members participating in the NAAQS review process, is to recognize the distinction between scientific evaluations and policy judgments. In my comments to Mr. Bill Wehrum and Dr. George Gray on improving the NAAQS review process, I noted—"It would be helpful if, at each step in the NAAQS process including each meeting of the scientists preparing the Criteria Documents and the Staff Paper and their review by CASAC, if each participant were reminded. "Every individual should recognize the distinction between scientific evaluation and policy decisions and recognize that the matters being dealt with are at the interface of science and policy. Each individual participant is asked to leave their individual ideologies and thoughts on policy decision outcomes at the door before deliberating on the science." This is not a matter of an individual's employment, i.e., academic, government, industry, etc. or political affiliation. It applies to all participants. This is an especially vexing issue for scientists involved in evaluating their own research results or that of close colleagues. In today's resource constrained world everyone wants to have their work used in the public arena, moreover, they would like to see the door left open or opened wider for them to do more work on the topic under consideration. Indeed, some individuals, including CASAC Panel Members, desire a "sense of accomplishment"—some individuals interpret that as—did we participate in lowering the NAAQSs' Some have suggested that there would be a "limited sense of accomplishment" if only the 24-hour $PM_{2.5}$ standard were lowered and the Annual $PM_{2.5}$ standard was left unchanged. Yes, scientific evaluations and policy decisions do get intertwined by individual scientists in expressing their own personal preferences on life science issues."

RESPONSE BY ROGER MCCLELLAN TO ADDITIONAL QUESTION FROM SENATOR VOINOVICH

Question 1. Dr. McClellan, as you heard Dr. Gray testify, he said characterization of uncertainties is extremely important. EPA is using a process called expert elicitation to respond to the NAS recommendation on this important issue. We will see the elicitation's results when the rule is finalized. Please elaborate on your concerns with this process.

Response. I am very familiar with the "expert elicitation" being used by the EPA to obtain expert opinions on the quantitative relationship between changes in $PM_{.5}$ concentrations in ambient air and changes in indices of adverse health effects. As I noted in my written testimony, I participated in a pilot expert elicitation study conducted by an EPA contractor as a prelude to the larger study recently conducted for the EPA.

In my professional opinion, the expert opinion elicitation process being used by the EPA is not a scientifically adequate approach to characterizing either the central estimate nor associated range of potential values for changes in adverse health outcomes associated with changes in ambient $PM_{2.5}$ concentrations for contemporary $PM_{2.5}$ levels found across the United States. The process is at best an elicitation of the opinions of a selected group of experts. It is not a substitute for rigorous scientific characterization of uncertainty of the kind I understand was recommended by the National Academy of Science/National Research Council in its 2002 report, "Estimating the Public Health Benefits of Proposed Air Regulations."

The outcome of the expert opinion elicitation process is influenced by five factors:

(1) the nature of the request from the sponsor, in this case the EPA, to the organization or individual(s) eliciting the opinions;
(2) the nature of the questions posed by the opinion elicitators;
(3) the manner in which the experts are identified and selected;
(4) the scientific background and personal biases of the experts; and
(5) how the individual opinions are compiled and reported.

It is apparent that a requested organization can influence the results by how they frame the question(s) presented for use in the elicitation process. In similar fashion the manner in which the questions are posed to the experts is critical. I understand that the central question asked, which was similar to that posed to me in the pilot study, was "What is the percent reduction in excess health risks associated with a 1 m^3 decrease in the ambient $PM_{2.5}$ concentration" This question assumes that there is a linear relationship between changes in $PM_{2.5}$ concentrations and excess health risks. This is an assumption, not a scientific fact. Indeed, I would argue that the results of the recent papers by Enstrom (2005) and Laden et al. (2006) clearly indicate a lack of a statistically

significant excess in mortality associated with contemporary ambient $PM_{2.5}$ levels and, thus, an absence of a linear exposure-response relationship at low levels of $PM_{2.5}$. Interestingly, this includes results from SouthernCalifornia and Stuebenville, OH, areas in which $PM_{2.5}$ levels were quite high in the past and have been substantially reduced.

The scientific background of the experts, their personal biases and how they are selected can influence the outcome of the elicitation process. Individuals whose research findings are under consideration in the expert opinion elicitation process are placed in an awkward position. Are they willing to set aside their own vested interest in seeing their results used in favor of giving a broader opinion? In giving an opinion, can they avoid being concerned with how the results will be used? Will a tighter standard result in a generally more favorable view of their research?

The manner in which results are ultimately compiled and presented is of critical importance. In the most recent process, the experts were polled individually and then brought together as a group to re-affirm the outcome. I argue that this consensus building approach suppresses uncertainty rather than contributing to a full expression of uncertainty. I would urge the EPA Administrator to not give significant weight to the results of the expert opinion elicitation process in setting the annual standard for $PM_{2.5}$. Moreover, I definitely feel it would be inappropriate to use the results of the expert elicitation process as input to the calculation of benefits in the Regulatory Impact Analysis.

Let me again emphasize that it is my professional opinion that it is imperative that the uncertainties associated with estimation of the excess adverse health effects of exposure to $PM_{2.5}$ at contemporary levels be rigorously characterized. In my opinion, the EPA at each step in the PM review process, failed to adequately characterize uncertainties. This was true of the Criteria Document, the Staff Paper and the Risk Assessment that under-girded the Staff Paper.

A rigorous uncertainty analyses would have started with critically examining the base data from critical studies and the very sophisticated statistical methods used. When the original published reports did not contain sufficient detail, the EPA should have obtained more detailed data and, if necessary, conduct additional analyses. This was done in a laudatory manner for some studies under the auspices of the Health Effects Institute, however, even more critical analysis would have been useful in better characterizing uncertainty in estimating $PM_{2.5}$ risks.

For example, very little was done to examine the validity of the Cox proportional hazard model for characterizing the very small estimated excess risks for $PM_{2.5}$ exposure and other confounders. Moolgavkar (2005 and 2006) has noted the limitations in this widely used model for estimating small excess risks. In none of the key studies being used to set the annual $PM_{2.5}$ standard were results presented for cigarette smoking, the major risk factor for cardiorespiratory disease. These results would have given an indication of how well the Cox model was working for the major risk factor, cigarette smoking, and, thus, gave some greater confidence in the use of the model for characterizing much lower levels of risk for $PM_{2.5}$. If historical information were not available on cigarette smoking this should have been identified as an uncertainty and, indeed, quantified.

The impact of cigarette smoking is apparent from the analysis of Enstrom (2005) using data for Southern California. Beyond presenting the analysis of $PM_{2.5}$ risks, the Enstrom paper also included the results of an analysis of the relative risk of death from all causes by cigarette smoking status. These results are of special value because they illustrate the substantial magnitude of the cigarette smoking effects relative to other risk factors such as air pollution. The baseline was never smokers (as of 1959 and 1972) for deaths 1973 to 2002 set at 1.000. Former smokers (as of 1959 and 1972) were Relative Risk (RR)-1.054 and increased to 1.253 for former smokers (as of 1972 only). Current smokers (as of 1972) had relative risks that increased with smoking intensity 1-9 cigarettes per day (cpd)RR-1.239; 10-19 cpdRR-1.97 cpdRR-1.871, 21-39 cpdRR-2.068 and 40+ cpdRR-2.543. The large relative risks related to cigarette smoking level provide perspective for the small relative risks reported for long-term $PM_{2.5}$ exposure. Indeed, in part because he had smoking histories available on the subjects in the Southern California cohort he was able to conclude These epidemiologic results do not support a current relationship between fine particulate pollution and total mortality in elderly Californians, but they do not rule out a small effect, particularly before 1988. For 1983-2002, the RR was 1.00. This included a substantial number of individuals exposed to $PM_{2.5}$ at concentrations above the current Annual Standard of 15 m^3. Moreover, the substantial effects of cigarette smoking emphasize the importance of accurate inclusion of cigarette smoking history in any long-term cohort study of the effects of PM and dictate that consideration of smoking be included in any quantitative characterization of uncertainty in estimating $PM_{2.5}$ risks.

Question 2. Dr. McClellan, you have been on these panels for a long time. Does the science ever point specifically to a number or are these standards really set in the judgment of the Administrator?

Response. I have served on each of EPA Clean Air Scientific Advisory Committees that have provided advice to the Administrator of the setting on revision of National Ambient Air Quality Standards for Particulate Matter. There has been a general acknowledgement by the Advisory Panel members that the science should inform the decisions on the four elements of the standard; (a) indicator, such as $PM_{2.5}$, (b) averaging time, such as annual, (c) numerical level, such as 15 m^3, and (d) the statistical form. There has also been general recognition that the Administrator has the ultimate responsibility for setting the standard using his/her judgment. The Advisory Panels have regularly reviewed and commented on the range of numerical levels presented in the Staff Paper, thereby acknowledging that the science can inform a broad range for setting the standard rather than the science yielding a specific numerical level. Each successive review of the Particulate Matter standard has become more contentious. In my opinion, some of the contentious nature has been driven by early concern by special interest groups and some members of the Panel as to the outcome of the process. In short, a premature view has developed that the $PM_{2.5}$ standard should be tightened, a view advanced long before the scientific evaluation had been completed. One way for CASAC to enhance the potential for the standard being tightened is to endorse a range of numerical values below that of the current standard. If this is done the judgment of the Administrator is constrained. This is exactly what was done when the CASAC PM Panel truncated the proposed range for setting an Annual $PM_{2.5}$ standard from 12 to 15 m^3 to 13-14 m^3. The CASAC PM Panel advanced an argument for changing the lower limit of the range from 12 to 13 m^3, i.e. uncertainty increased below 13 m^3. In none of the letters from the CASAC Chair to the Administrator was a rigorous rationale provided for reducing

the upper bound of the range from 15 to 14 m^3. In my view, by endorsing an exceptionally narrow range of 13 to 14 m^3 the CASAC PM Panel ignored the uncertainty in the science and attempted, inappropriately in my view, to exercise some of the judgment that is reserved for the Administrator by the Clean Air Act. The CAA wisely calls for CASAC to advise the Administrator and for the Administrator to use judgment in setting the standard. The CASAC PM Panel, in narrowing the range to 13 to 14 m^3, stopped one small step short of attempting to set the $PM_{2.5}$ Annual standard.

Question 3. As a member of the CASAC panel was there complete agreement on the recommendations.

Response. There was not complete agreement among CASAC PM Panel Members on the narrowing of the range for the Annual $PM_{2.5}$ standard from 12 to 15 G53 to 13 to 14 m^3. George Wolff and I, who had both previously served as Chair of the Clean Air Scientific Advisory Committee, disagreed with the proposal. It is of interest that the decision to narrow the range was reached during short conference calls of the Panel and by electronic exchanges among small groups of members. The decision was not one reached on the basis of a typical face-to-face public meeting of the Panel. There was also intense pressure to obtain and present a consensus view and to provide a letter from CASAC to the Administrator that was devoid of attached individual views as customary for CASAC letters to the Administrator, especially on important matters. I exercised my rights as a private citizen to prepare a letter to the docket expressing my views on the setting of the PM standard. In my professional opinion, the available scientific information is consistent with setting an Annual $PM_{.5}$ Standard in the range of 12 to 15 m^3 as articulated in the EPA Staff Paper with the specific numerical level to be selected by the Administrator based on judgment as specified in the Clean Air Act.

PREPARED STATEMENT OF ANNE SMITH, VICE PRESIDENT, CRA INTERNATIONAL

Mr. Chairman and members of the committee, thank you for your invitation to participate in today's hearing. I am Anne Smith, and I am a Vice President of CRA International. I am a specialist in environmental risk assessment and integrated assessment to support environmental policy decisions, which was a core element of my Ph.D. thesis at Stanford University in economics and decision sciences. I have performed work in the area of risk assessment over the past 30 years, including as an economist in the USEPA's Office of Policy, Planning, and Evaluation, as a consultant to the USEPA Air Office, and in many consulting engagements since then for Government and private sector clients globally while employed first at Decision Focus Incorporated and then CRA International. I have also served as a member of several committees of the National Academy of Sciences focusing on risk assessment and risk-based decision making.

I have been deeply involved in assessment of the evidence on risks from ambient fine particulate matter ($PM_{2.5}$) since EPA first turned to the task of identifying an appropriate National Ambient Air Quality Standard (NAAQS) for $PM_{2.5}$ over 10 years ago. I testified to this same committee in 1997 on the nature of the scientific evidence underlying the $PM_{2.5}$ NAAQS proposed at that time. I thank you for the opportunity to share my perspective today on the current scientific evidence and associated risk assessment for $PM_{2.5}$ and how it has evolved since 1997. My written and oral testimony today provide a statement of my own research and opinions, and does not represent a position of my company, CRA International.

I would like to start by summarizing what I think are the most important and overarching considerations that should be accounted for when considering whether to alter the current $PM_{2.5}$ NAAQS, which include an annual average limit of 15 m^3 and a 24-hour average limit where the 98th percentile of observations over all days must be below 65 m^3. I will then summarize results of analyses I have done to synthesize the recent $PM_{2.5}$ health studies into an assessment of risks. Complete details and documentation of my analyses are in my written comments on the current Proposed Rule for a revised $PM_{2.5}$ NAAQS, which were submitted into the $PM_{2.5}$ docket in April, 2006. I am attaching a copy of my written comments to EPA to further substantiate the points that I make in my testimony today.

The key points that I wish to make about the scientific evidence on risks of $PM_{2.5}$ that are relevant for making a decision on the standard are:

• EPA and the courts recognize that the $PM_{2.5}$ NAAQS must be set at a level that still has some positive level of risk, because the science has yet to advance far enough to identify any threshold exposure level for effects, below which risk would be indistinguishable from zero. This was true in 1997 and it remains true today.

• EPA's own quantitative estimates of mortality risk at attainment of the current NAAQS are lower today than they were when EPA set that standard in 1997 "with an adequate margin of safety," after accounting for the many uncertainties. This is true for both long-term ("chronic") exposures to $PM_{2.5}$ (which are addressed by the annual average limit) and short-term ("acute") exposures (which are addressed by the 24-hour average limit).

• The reduction in the quantitative estimates of risk is apparent even in EPA's own risk analysis, but most of the reasonable alternative results reported in the same studies that EPA has relied on imply even lower quantitative risk estimates for $PM_{2.5}$.

• Looking more broadly beyond quantitative risk estimates, the many additional studies of $PM_{2.5}$ mortality risks since 1997 have demonstrated that many of the risk estimates become "statistically insignificant" when re-estimated in reasonable alternative ways. A "statistically insignificant" result directly implies a positive probability that there is no effect at all. Thus, when we look at all of the data in the new studies as a group, we find more statistical evidence now than was available in 1997 that $PM_{2.5}$ may not be the culprit pollutant, and that there may be no causal relationship at all between $PM_{2.5}$ and mortality.

In thinking about whether to tighten either the annual or daily standard, one might ask, what has changed in our knowledge since 1997 that would undermine the Administrator's 1997 judgment that the current $PM_{2.5}$ NAAQS are neither more nor less stringent than necessary to protect the public health with an adequate margin of safety? A thorough review of the new evidence suggests that the margin of safety that the Administrator selected in 1997 is likely to be larger than was thought at the time.

QUANTITATIVE ESTIMATES OF RISK REMAINING AT THE CURRENT STANDARD HAVE FALLEN

EPA has acknowledged that the $PM_{2.5}$ NAAQS cannot be set at a level that corresponds to zero risk.[1] However, EPA has also argued that its quantitative risk estimates cannot be used to identify a specific point where it should set a standard:

"[I]n the Administrator's view, a risk assessment based on studies that do not resolve the issue of a threshold is inherently limited as a basis for standard setting, since it will necessarily predict that ever lower standards result in ever lower risks, which has the effect of masking the increasing uncertainty inherent as lower levels are considered. As a result, while the Administrator views the risk assessment as providing supporting evidence for the conclusion that there is a need to revise the current suite of $PM_{2.5}$ standards, he judges that it does not provide a reliable basis to determine what specific quantitative revisions are appropriate."[2]

I concur that a risk assessment that makes no attempt to incorporate the uncertainty on where a threshold may exist will indeed only serve to promote ever lower standards without a sound basis. Since EPA has not incorporated such uncertainty into its risk assessment, that risk assessment is indeed incapable of helping to identify where to set the standard. However, since EPA views the risk assessment as supporting a conclusion on whether there is a need to revise the standard, it is appropriate and relevant to compare EPA's current quantitative risk estimates and the associated statistical measures of a $PM_{2.5}$ effect to those estimates that were available in 1997. In the Proposed Rule, EPA partially acknowledges that risk estimates are lower today than in 1997 for the two cities that were included in both its 1997 and current risk analyses. With respect to short-term exposure risk estimates, EPA states that "the magnitude of the estimates associated with just meeting the current annual standard... is similar in one of the locations. . . and the current estimate is lower in the other location." [3] With respect to the long-term exposure risks, EPA states that the risk estimates "are very similar for the two specific locations included in both the prior and current assessments."[4]

EPA does not provide the actual numerical estimates for these two cities. They are:

• For acute risks in Los Angeles, in 1997 EPA estimated that 1.7 percent of mortality would continue to be attributable to $PM_{2.5}$ once Los Angeles would be in attainment with the current NAAQS. Today EPA's risk estimate has fallen to 0.5 percent and this current estimate is statistically insignificant (which means that there

[1] See, for example, the Proposed Rule, p. 2622 (i.e., 71 FR 2622).
[2] 71 FR 2648.
[3] 71 FR 2640.
[4] 71 FR 2640.

is a fairly large chance that this particular estimate suggests that there is really no $PM_{2.5}$ effect at all).

• For acute risks in Philadelphia, in 1997 EPA estimated that 1.5 percent of mortality would continue to be attributable to $PM_{2.5}$ at attainment of the current NAAQS. The risk estimate that EPA now uses for Philadelphia is 2.2 percent. Although this is higher than in 1997, EPA has selected a single estimate out of a very large number of estimates reported in the epidemiological study it is relying on for Philadelphia. In fact, that study actually concluded that $PM_{2.5}$ did not appear to explain the mortality risk as well as ozone, and the residual risk for $PM_{2.5}$ after simultaneously accounting for the role of ozone would have produced a lower estimate—about 0.8 percent—which is lower than in 1997. This more thoroughly-controlled estimate also is not statistically significant.

• Chronic risk estimates do not vary from city to city, because the statistical method to estimate relative chronic risks produces a single value that applies to all cities. I will therefore only relate the results for Los Angeles here. For chronic risks, in 1997 EPA estimated that 2.0 percent of mortality would continue to be attributable to $PM_{2.5}$ at attainment of the current NAAQS. Today, EPA's risk estimate for the same attainment status is 1.8 percent—in other words, the chronic risk estimate also is lower now, even though the quote from the Proposed Rule above suggests that the estimate has not changed.

The Proposed Rule only referred to a comparison of risks for these two cities. However, it is actually possible to make the same comparison for the other six cities that EPA has included in its current risk analysis. This is because there was only one $PM_{2.5}$ acute mortality study it could have used for each of those cities back in 1997 the same one that it used for Los Angeles and Philadelphia.[5] For five of the other six cities in the current risk analysis, EPA's acute risk estimates today are lower than they would have been estimated to be in 1997, and all the cities have lower chronic risk estimates. Table 1 summarizes the cities and the results of my comparison of their risk estimates.

When I reviewed the original papers that EPA is relying on, I also found that EPA's risk analysis has selectively used the highest or near-highest risk estimates supported by each paper. This means that risks estimates that more fully reflect the body of evidence are likely lower still than EPA's risk analysis suggests. Additionally, as for Philadelphia, I found that San Jose would have had a much lower risk estimate than in 1997—literally zero now—if EPA had chosen to use the one reasonable alternative result for $PM_{2.5}$ reported in the San Jose study.[6] Thus, the full body of evidence can support risk estimates that would be lower now than in 1997 for every one of the eight cities in EPA's current risk analysis.

[5] This was the paper by Schwartz, Dockery, and Neas (1996) on acute risks in six U.S. cities.

[6] That is, EPA's risk estimate for San Jose is based on a 1-pollutant regression that associated mortality with $PM_{2.5}$ on the same day as death. The study also reported results of a comparable 1-pollutant regression that was identical in all ways except that it associated mortality with $PM_{2.5}$ from the day before death. The latter regression produced a negative risk estimate, which I interpret to be evidence of no effect at all (rather than evidence of a beneficial effect of $PM_{2.5}$).

Table 1. Comparison of EPA's Risk Estimates for Attainment of the Current Standard Now Versus in 1997.

		Has EPA's risk estimate gone up or down since 1997?	Is statistical significance of estimate robust to alternative model choices?
Acute risk	Philadelphia	Up	Not robust
	Los Angeles	Down	Not robust
	Phoenix (*)	Down	Not robust
	St. Louis (*)	Down	Not robust
	Boston (*)	Down	Not robust
	Detroit (*)	Down	Not robust
	Pittsburgh (*)	Down	Not robust
	San Jose (*)	Up	Not robust
Chronic risk	All Cities	Down	Not robust

(*) Although this city was not in the 1997 Risk Analysis, it is possible to determine what risk would have been estimated for each, using the only data that were available in 1997, following EPA's same decisions for Philadelphia and Los Angeles. Specifically, the 1997 risk estimate is based on the "combined" estimate in Schwartz, Dockery, and Neas (1996) for all cities except for Boston and St. Louis, for which it is the city-specific estimate in that paper.

Table 1 also reports that the $PM_{2.5}$ findings are not statistically significant across all of the alternative reasonable risk estimates in each underlying study. This was not the case in 1997. At that time, there was a much more limited set of studies and estimates within each study—but for some cities, all the estimates available at the time were statistically significant. Today, the opposite it true. Every single study that EPA has relied on for its current risk analysis contains alternative estimates that indicate that $PM_{2.5}$ does not have a statistically significant association with mortality, yet EPA chose not to use this part of the new information.

In conclusion, EPA has stated that the risk assessment's role is to provide "supporting evidence" on whether there is a need to revise the $PM_{2.5}$ standard. In this role, EPA's own risk analysis provides no evidence supporting a decision to tighten the standard now. The risks are lower now than they were when the standard was set in 1997. The higher estimates of risks were determined to be "requisite to protect the public health with an adequate margin of safety" in 1997, and the quantitative risk analysis suggests that that margin of safety has grown, not narrowed, as a result of the many more recent $PM_{2.5}$ health effects studies.

The question then remains whether other aspects of the new evidence provide an overriding reason for tightening the standard. The other part of EPA's reasoning for how to set the standard relies on what EPA calls an "evidence-based approach." Simply put, EPA looks at all of the studies that estimate the statistical relationship of $PM_{2.5}$ with health effects, and seeks to identify a level of $PM_{2.5}$ above which statistically significant effects are found, and below which statistically significant effects are not found.

In applying the evidence-based approach, EPA states that the large quantity of new studies of acute effects justifies the use of acute studies to set the 24-hour standard, and that chronic studies should be used to determine where to set the annual standard:

"Given the extensive body of new evidence based specifically on $PM_{2.5}$ that is now available, and the resulting broader approach presented in the Staff Paper, the Administrator considers it appropriate to use a different approach from that used in the last review to select appropriate standard levels. More specifically, the Administrator's proposal relies on an evidence-based approach that considers the much expanded body of evidence from short-term exposure $PM_{2.5}$ studies as the principal basis for selecting the level of the 24-hour standard and the stronger and more robust body of evidence from the long-term exposure $PM_{2.5}$ studies as the principal basis for selecting the level of the annual standard."[7]

I will next discuss how the evidence in the long-term exposure studies of $PM_{2.5}$ has weakened since 1997, thus removing any necessity to tighten the annual stand-

[7] 71 FR 2648.

ard under EPA's evidence-based approach. I will then discuss how the evidence in the short-term exposure studies of $PM_{2.5}$ that are the basis for the 24-hour standard also has weakened.

THE EVIDENCE IN LONG-TERM EXPOSURE STUDIES HAS WEAKENED SINCE 1997

In 1997, the two prominent long-term exposure studies (one based on a sample population, or "cohort" in 154 U.S. cities that was tracked by the American Cancer Society, and one based on a sample population in just six US cities that was tracked by Harvard School of Public Health) both had published findings of a statistically significant relationship between long-term exposure to $PM_{2.5}$ and life expectancy. These studies were subjected to an extensive process of reanalysis under the auspices of the Health Effects Institute (HEI) that was released in 2000.[8] This reanalysis is widely reputed to have confirmed the original studies' results; however, a complete reading of the actual report shows that some major statistical concerns underlying those results were unearthed. Although a positive $PM_{2.5}$ effect was still found in those data sets, the ability to interpret those results as clearly causal in nature was weakened.

EPA acknowledges that the concerns identified in the HEI reanalyses of the long-term exposure studies for $PM_{2.5}$ remain unresolved to the present time:

The Administrator also recognizes a contrasting view as to the interpretation of and weight to be accorded to the results from the ACS-based studies (Pope et al., 1995; Krewski et al., 2000; Pope et al., 2002). In this view, the ACS-based studies are not sufficiently robust to support a policy response that would tighten the annual $PM_{2.5}$ standard based on the evidence. This view emphasizes the sensitivity of the results of these studies to plausible changes in model specification with regard to accounting for the geographical proximity of cities and the correlation of air pollutant concentrations within a region, effect modification by education level, and inclusion of SO_2 in the model. In this view, these sensitivities suggest potential confounding or effect modification that has not been taken into account. For example, concern has been raised about the sensitivity of results in the reanalysis of data from the ACS cohort study (Krewski et al., 2000) to inclusion of SO_2 in the models. [T]he reanalysis found that $PM_{2.5}$, sulfates, and SO_2 were each associated with mortality in single-pollutant models. However, in two-pollutant models with SO_2 and $PM_{2.5}$, the relative risk for $PM_{2.5}$ was substantially smaller and no longer statistically significant, whereas the effect estimates for SO_2 were not sensitive to inclusion of $PM_{2.5}$ or sulfates in two-pollutant models. In this view, the ACS-based risk estimates are more robust for SO_2 than for $PM_{2.5}$ or sulfates. In further extended analyses, Pope et al. (2002) reported that effect estimates were not highly sensitive to spatial smoothing approaches intended to address spatial autocorrelation, while findings of effect modification by education level were reaffirmed. Results of multi-pollutant models were not reported by Pope et al. (2002). Because the correlation coefficient between $PM_{2.5}$ and SO_2 was 0.50 in the ACS data, in this view it is plausible to believe that the independent effects of the two pollutants could be disentangled with additional study."[9]

The quote above is lengthy, which highlights that the concerns identified in the long-term exposure studies are many. The quote above also indicates that the new set of results using the American

Cancer Society cohort that was published after the HEI reanalyses (i.e., Pope et al., 2002) did not help resolve these issues. Specifically, the 2002 paper ignores concerns that the purported $PM_{2.5}$ effect instead might be attributable to the gaseous pollutant SO_2, and re-affirms a troubling finding that $PM_{2.5}$ only seems to create mortality risk only for individuals who have not continued their education beyond the high school level. (The latter finding is discussed further below.) Additionally, the 2002 paper still finds that the $PM_{2.5}$ effect is diminished and rendered insignificant when applying statistical methods to correct a clear statistical error that the HEI report found in the original results. Nevertheless, the Pope et al. (2002) paper continues to use the estimation method that is subject to error except in a sensitivity analysis; and EPA continues to rely on the uncorrected estimates in its risk analysis. Even with these dubious selections from the full body of literature, EPA's estimates of long-term exposure risk are lower than in 1997.

Figure 1 illustrates the degree to which the evidence on long-term exposure risk has fallen, both in the overall magnitude of the risk estimate, and also in terms of a greater degree of uncertainty in the estimate. Figure 1 uses the case of Los Angeles at attainment of the current standard, yet the relative patterns evident in this

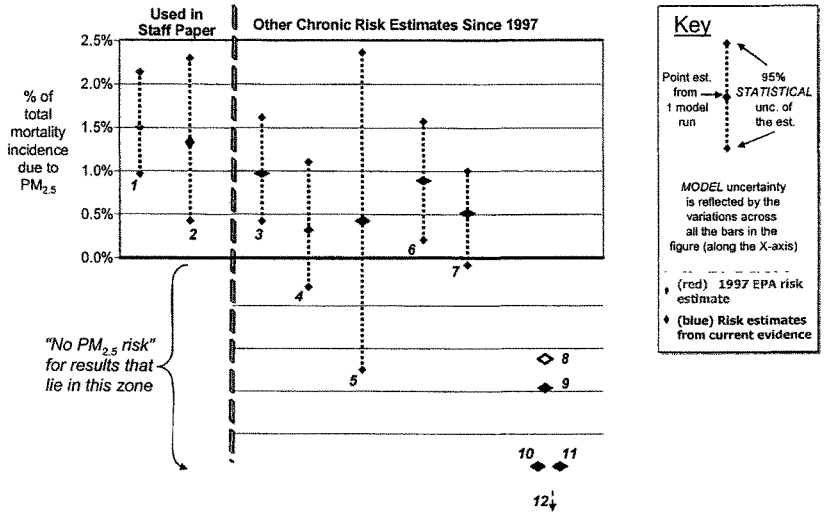
[8] The report on findings of these reanalyses is Krewski et al. (2000).
[9] 71 FR 2652.

figure are the same for all cities in the U.S. All of the risk estimates in Figure 1 labeled "1" through "7" are based on the American Cancer Society cohort, which has received the majority of attention. The estimate on the far left of the figure, labeled "1" is the estimate from 1997 (note that the estimate is 1.5 percent, as reported for Los Angeles in the preceding section), and the estimates to the right are other key results from the HEI reanalyses and from the more recent Pope et al. (2002) paper. The estimate labeled "2" is the single result from the many new estimates that is used for the current EPA estimates of long-term risk (which is 1.3 percent, as I stated in the previous section).

It is quite apparent from the figure that the current risk estimate is among the highest that could be found among the more recent results. If any of the others (labeled "3" through "7") had been used for EPA's risk analysis, the current risk estimate of 1.3 percent for Los Angeles would instead be in the range of 0.3 percent to 1.0 percent—much lower than the original 1997 risk estimate that was available when the current standard was first set.

The set of results on the far right of the figure (labeled "8" through "12") reflect the findings based on a new study of a third sample population that had not been identified or studied as of 1997. It is known as the "Veterans' Cohort." I believe this study to be of some policy relevance regarding whether or not the annual standard needs to be tightened, given that this study finds no effect at all of $PM_{2.5}$ on life expectancy in this particular cohort. EPA has chosen to give "greatest weight" to results from the American Cancer Society and the Six Cities cohorts because they have been reanalyzed and scrutinized so thoroughly. While this may be a reasonable judgment, EPA has actually gone further than that, and accorded the Veterans' Cohort results zero weight. Its findings should be acknowledged with somewhat more than zero weight. When one does so, the overall evidence regarding long-term $PM_{2.5}$ risks is further weakened.

Figure 1. Evidence of the Eroding Strength of Association in Long-Term $PM_{2.5}$ Risk Studies
(estimates are for Los Angeles at attainment of the current standard)

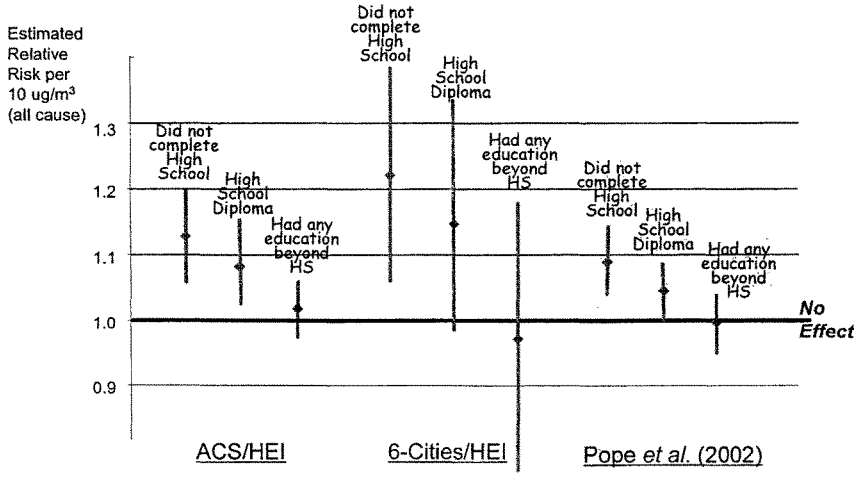


1. Pope *et al.* (1995), Table 3, row 1, column 4 – all combined, all cause mortality, for $PM_{2.5}$
2. Pope *et al.* (2002) Table 2, row 1, column 3 – single pollutant + "all covariates" using "averaged" PM
3. Krewski *et al.* (2000), Table ES-4 – row 3 column 1 (single pollutant)
4. Krewski *et al.* (2000), Table 1, row 18, column 4 – 2-P case (ecologic controls including SO_2)
5. Krewski *et al.* (2000), Table ES-6, row 5 – with "25%" ecologic covariates using regional adjustment
6. Pope *et al.* (2002), Table 2, row 1, column 1 – single pollutant + "all covariates" using 1979-1983 PM
7. Pope *et al.* (2002), Figure 3A – single pollutant + "highest P-value" spatial smoothing using 1979-1983 PM
8. Lipfert *et al.* (2000b), Table 7, deaths in 1976-81 associated with PM2.5 data from 1979-1981 (coeff=-5.28)
9. Lipfert *et al.* (2000b), Table 7, deaths in 1982-1988 associated with PM2.5 data from 1982-1984 (coeff= -6.11)
10. Lipfert *et al.* (2000b), Table 7, deaths in 1982-1988 associated with PM2.5 data from 1979-1981 (coeff= -10.07)
11. Lipfert *et al.* (2000b), Table 7, deaths after 1988 associated with PM2.5 data from 1982-1984 (coeff= -10.78
12. Lipfert *et al.* (2000b), Table 7, deaths after 1988 associated with PM2.5 data from 1979-1981 (coeff=-15.35

EPA also refers to the perplexing finding that level of education determines whether or not there is a $PM_{2.5}$ association. Figure 2 illustrates this finding, which was first identified in the HEI reanalyses, and which remains in the more recent Pope et al. (2002) study. Clearly education *per se* is not believed to be the cause of sensitivity to exposure to $PM_{2.5}$, yet the important (and still unanswered) question is: what is educational level indicating about risks that these sample populations face? What could possibly explain the complete lack of a $PM_{2.5}$ effect among those with higher educations? When such a pattern appears in epidemiological study results, it indicates that there is still an important explanatory factor that is missing from the statistical estimation method something correlated with education. Until that factor is identified and included in the estimation of $PM_{2.5}$ risks, estimates of the effect of $PM_{2.5}$ are biased. The $PM_{2.5}$ estimate could be higher, or it could vanish altogether. Thus, the unexplained pattern related to education in all of these studies remains a very important warning about the pitfalls of making a causal interpretation regarding long-term exposure risks of $PM_{2.5}$.

In summary, the evidence against a need to tighten the annual standard is not just founded on the fact that the numerical long-term risk estimates are now lower than when the current standard was set. The more important point is that the basis for interpreting the long-term studies as unbiased evidence of a causal relationship between $PM_{2.5}$ and chronic mortality risk has weakened. This was a concern in 1997, and the reanalyses and new studies since then have done more to amplify these concerns than to allay them. In the face of this evidence of greater uncertainty, combined with the reduced quantitative risk estimates, there is no justification for tightening the annual standard on the basis of the long-term exposure studies.

Figure 2. New Analyses of Long-Term Exposures Risks Since 1997 Find that Educational Level Is an Important Determinant of Whether PM2.5 is Associated with Mortality
(Sources: "ACS/HEI" and "6-Cities/HEI" are based on numerical results reported in Krewski *et al.* (2000), Summary Table 3. Results cited from Pope *et al.* (2002) are in Figure 4A of that paper.)



A Summary CASAC's Case for Tightening the Annual Standard

EPA's Clean Air Scientific Advisory Committee (CASAC) has made the case to tighten the annual standard with two lines of reasoning, neither of which is founded on the long-term risk studies.[10]

CASAC's first line of reasoning is that EPA reports substantial risk would remain at the current standard. As EPA and the courts have long established, the $PM_{2.5}$ NAAQS cannot be a zero-risk standard. CASAC was concerned by the estimates of remaining risk, but never deliberated the question of whether this risk estimate had risen or fallen since the standard was deemed "requisite to protect the public health with an adequate margin of safety." As I have shown above, the risk estimates fell, both for chronic and acute risks, but EPA never reported this fact to CASAC during

[10] CASAC's reasoning is stated in a letter to the Administrator on March, 21 2006, pp. 3-4.

CASAC's review of the Staff Paper and associated risk assessment. In the face of this fact, the only other argument to tighten the annual standard might be if there were stronger reason to believe that the effects found in these studies are causal in nature. However, EPA set the current standards with a presumption (precautionary in nature) that the estimated $PM_{2.5}$ risks were causal. This cannot therefore be the rationale to tighten the standards.

Hence, to argue that the standard should be tightened because there is evidence that risk remains at the current standard is a logic that would force a tightening of the standard in every future review cycle, even if no new evidence were to have become available at all since the previous review. There is nothing in the law or in precedent that dictates that the standard has to be tightened as the result of a NAAQS review.

CASAC's second line of reasoning comes closer to the heart of how EPA first set the standard. CASAC notes that there are three new acute studies that find $PM_{2.5}$ associations with mortality at annual averages below the current annual standard (all with reported annual averages in the range of 13 to 14 m^3). These studies are: Burnett and Goldberg (2003) for 8 Canadian cities combined; Mar et al. (2003) for Phoenix, and Fairley (2003) for Santa Clara County, CA (referred to in the risk analysis as San Jose).[11]

The first thing to realize about this part of CASAC's case for a tightened annual standard is that it is using studies that consider only how day-to-day changes in $PM_{2.5}$ levels affect day-to-day numbers of deaths relative to the number of deaths that might otherwise be expected on each day (e.g., relative to numbers of deaths that are expected to occur on each day based on established patterns related to the time of year, time of week, weather, etc.). Such acute effects of a pollutant are generally believed to be associated with spikes in $PM_{2.5}$, although studies to date rarely report evidence of any threshold level below which the association disappears. Nevertheless, there is no clear linkage between the annual average in a city, and the extent to which day-to-day spikes in $PM_{2.5}$ might be occurring. If an acute effect is found in a city that happens to have a low annual average, there is no reason to believe that the estimated association is not still due to sudden upward changes in $PM_{2.5}$ from one day to the next. The city may simply have a large number of very clean days that pull the annual average $PM_{2.5}$ down, while not eliminating the presence of many days of sudden increases in pollution that are logically likely to be the cause of any acute risk that the study is finding.

Thus, it is not necessarily correct to assume that if acute risks are found in a city with a low annual average pollution, then such risks exist in all cities with low annual average pollution. In fact, if one believes that there must be a threshold where the acute risk from exposure to pollution drops off somewhere above zero, then the linkage between the annual average of pollution and existence of acute risk is not only unclear, but illogical. For reasons such as these, EPA has decided to use the plethora of acute risk studies now available to set the 24-hour standard that that type of study more meaningfully informs. EPA has decided not to use acute studies to set an annual standard.[12]

Nevertheless, even if one were to use acute effects studies to determine an "adequate margin of safety" for lower levels of long-term exposure to pollution, there are good reasons to believe that the annual average $PM_{2.5}$ reported for each of the three studies cited by CASAC may not be a good indicator of the long-term exposure levels that account for the risk findings in these studies. I explain why for each of the three:

Goldberg and Burnett (2003). This study reports a $PM_{2.5}$ association for eight Canadian cities combined. The annual average of 13.3 m^3 is an average over all of the eight cities, while the annual averages in the individual cities vary from 9.5 m^3 to 17.7 m^3. There are no city-specific results reported to help indicate whether the estimate of an acute effect is due to effects in each of the eight cities, or only in a few.[13] Evidence in the paper suggests that there may in fact be different effects in each city.

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[11] CASAC actually cites a paper by Lipsett et al. (1997) in its letter, but that paper has nothing to do with $PM_{2.5}$ mortality. I interpret CASAC to have wanted to cite Fairley (2003).

[12] See the quote on p. 5 above, taken from 71 FR 2648.

[13] The other multi-city $PM_{2.5}$ mortality studies (based on the Six Cities data set) report effects by individual city as well as for the combined set. This was the data on which EPA set the current standards, and in doing so, EPA used annual averages for only the individual cities that did have significant effects within the set of six. The lowest such city was Boston, with an annual average of 15.6 m^3, which was the basis for the current annual average standard of 15 m^3.

Another concern with this study is that it is a reanalysis of a more comprehensive study that included consideration of the role of gaseous pollutants as well.[14] The original study concluded that the gaseous pollutants had a much greater ability to explain mortality risks

than both $PM_{2.5}$ and $PM_{10-2.5}$ combined. However, when the paper had to be reanalyzed, the authors did not reanalyze the portions that considered gaseous pollutants in conjunction with particulate pollution, and so this finding is no longer discussed.

Fairley (2003). This study used data from Santa Clara County, CA, over a seven year period, and during that time pollution levels were falling dramatically. Although the annual average $PM_{2.5}$ that is attributed to this study is 13.6 m^3, the annual average was as high as 18.4 m^3 at the start, and fell progressively to 9.5 m^3 by the end of the 7 years studied.[15] Peak levels of $PM_{2.5}$ were also falling, starting at a 98th percentile of 88 m^3 for the first year and ending at 25 m^3. Such a wide range within this one city's data set begs the question: are the reported acute effects relationship driven largely by the high levels in the early years, or are they also evident in the later years? This highly relevant question is never mentioned, let alone analyzed, by the authors. Lacking any exploration of such an obviously relevant issue, it would seem a dubious proposition to use the annual average over the entire time period in this one study as the basis for a national ambient standard.

Another concern with this study is that it reports $PM_{2.5}$ risk estimates for two alternative methods of estimation, both of which are reasonable. One method considers whether deaths tend to fluctuate with the same day's $PM_{2.5}$ levels and the other method considers whether deaths tend to fluctuate with the previous day's $PM_{2.5}$ levels. The same-day estimate finds the positive association that this study is known for, but the estimate based on $PM_{2.5}$ on just the previous day is actually in the negative direction. Complete reversal of evidence of a $PM_{2.5}$ mortality effect by considering $PM_{2.5}$ levels only 24 hours apart in time presents a concern for interpreting the study's same-day estimate as a causal one. However, there is no discussion of what these conflicting results might mean.

Mar et al. (2003). This study considered acute risks in Phoenix, AZ, with annual average $PM_{2.5}$ levels of 13.5 m^3. There are 10 estimates of $PM_{2.5}$ risk in the paper, and only 3 of them are significant. More importantly, this is not the only paper that studied the ability of this same set of $PM_{2.5}$ data to explain acute mortality risks in Phoenix. One of the other studies found that $PM_{2.5}$ did not have any explanatory power, and found instead that the coarse fraction of PM had explanatory power.[16] The third study found evidence that there is a threshold below which $PM_{2.5}$'s apparent ability to explain changes in daily mortality disappeared.[17] That threshold appeared to be above 20 m^3. If there is a threshold, then the rationale for a linkage between annual average $PM_{2.5}$ and acute risks simply falls apart.

A final concern with all three of the Phoenix studies is that none of them considered whether the $PM_{2.5}$ effect would remain if pollutants such as CO, SO_2, ozone, or NO_2 were also included in the analysis. This is a critical gap in many of the current studies because the new body of papers on $PM_{2.5}$ health effects reveals that $PM_{2.5}$ effects usually disappear when one of the gaseous pollutants is explored. This is addressed in the next part of my testimony.

In summary, CASAC makes its case to tighten the annual standard on the basis of acute, not chronic effects studies. There are logical problems with this approach to setting an annual standard; these logical problems become apparent when looking at each of the three acute studies that CASAC cites as its basis for recommending an annual standard that is tighter than the current one.

THE STATISTICAL EVIDENCE ON ACUTE EFFECTS OF $PM_{2.5}$ HAS ALSO WEAKENED SINCE 1997.

In 1997, there existed only one study that had used actual measurements of $PM_{2.5}$ and estimated whether daily numbers of deaths might be associated with day-to-day variations in the $PM_{2.5}$. This was a study using the data from the Harvard study of six U.S. cities reported in Schwartz et al. (1996), and it was used as the basis for the current standards. In that study, statistically significant associations of $PM_{2.5}$ and acute mortality were found in three of the four cities with the highest 98th percentile $PM_{2.5}$ levels, which ranged from 42 m^3 to 44 m^3. The city with the highest $PM_{2.5}$ 98th percentile (which was 82 mG53) did not produce a statistically

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[14] The original paper was Burnett et al. (2000)

[15] To know this, one must go back and read the original study that this is a reanalysis of, Fairley (1999).

[16] Clyde et al. (2000).

[17] Smith et al., (2000)

significant association, nor did the two cities with the lowest 98th percentile levels of 32 and 34 m^3. This was the best available information at the time,. Other than the anomaly for the city with the highest $PM_{2.5}$ exposures, it did at least suggest that there might be a range above which effects were more likely and below which they were more unlikely.

While this study was used as the primary basis for the current standards (including the annual standard), there were many concerns expressed with uncertainties in the estimation methods. In particular, there was concern that this study had not considered the explanatory role of any of the other common pollutants like CO, SO_2, ozone, and NO_2. (These are often called the "gaseous pollutants" because that distinguishes them from various forms of particulate pollutants that are regulated under the PM NAAQS.) It was argued that $PM_{2.5}$ might be simply playing a proxy role for a gaseous pollutant also present in the air in these cities.

As new acute $PM_{2.5}$ studies were performed after 1997, a number of these studies did strive to explore the respective roles of $PM_{2.5}$ and gaseous pollutants in the observed statistical associations. This was done by using "2-pollutant" or "multi-pollutant" methods, as contrasted to the "1-pollutant" method that only allows a single pollutant (e.g., $PM_{2.5}$ in this case) to have any opportunity to explain mortality risk. One of the little recognized but important insights of this body of studies is that when gaseous pollutants also have been considered in a study, the gaseous pollutant has taken over the explanatory role from $PM_{2.5}$ in a majority of the cases.

I determined this in my review of the studies since 1997. Specifically, I attempted to identify all of the $PM_{2.5}$ health effects studies cited in the Criteria Document (including both mortality and morbidity effects studies) that had reported results of any estimates for $PM_{2.5}$ using a 2-pollutant method of estimation for at least one gaseous pollutant. I found 10 such papers among all the new studies that did report a statistically significant association for $PM_{2.5}$. Of these 10, 8 saw $PM_{2.5}$ lose its ability to explain mortality risk when studied using a 2-pollutant method. (In the other two studies, both the $PM_{2.5}$ and gaseous pollutant retained statistical explanatory power.)

Often it is suggested that 2-pollutant methods are not useful because it is impossible to unravel the effects of two pollutants that both move up and down together in near synchrony (i.e., they are highly "correlated"). However, my review of these papers did not find evidence that this was a problem. If it were a problem, then both the $PM_{2.5}$ and gaseous pollutant would lose their explanatory power. What I found instead was that in seven of the eight studies where $PM_{2.5}$ lost its erstwhile explanatory power when it was the only pollutant considered, the gaseous pollutant retained its explanatory power. Otherwise stated, of the ten studies that I started with, only one seemed to be affected by intractable statistical problems making it impossible to unravel the separate effects of the two pollutants.

These papers are summarized in Table 2, which is more fully explained in my written comments to EPA of April 2006, which I am submitting with this testimony.

Table 2. Evidence from Recent Studies that Acute $PM_{2.5}$ Effect Estimate Is Often Lost When Estimated in a 2-Pollutant Model with a Gaseous Co-Pollutant

Paper	City	Effect Estimated	Was any $PM_{2.5}$ Coefficient Significant?		Gaseous Pollutant Signif in 2-P?	Gaseous Pollutant Included
			1-P	2-P		
Delfino *et al.*, 1997	Montreal	ER visits	Yes	No	Yes	O_3
Sheppard, 2003	Seattle	Hosp adm	Yes	No (**)	Yes	CO
Lipfert *et al.*, 2000a	Philadelphia	Mortality	Yes	No	Yes	O_3
Korrick *et al.*, 1998	NH Mtns	Lung function indicators	Yes	No	No	O_3
Thurston *et al.*, 1994	Toronto	Hosp adm	Yes	No	Yes	O_3
Moolgavkar, 2003	Los Angeles	Hosp adm	Yes	No	Yes	CO, NO_2
		Mortality	Yes	No	Yes	CO
Delfino *et al.*, 1998	Montreal	ER visits	Yes	No	Yes	O_3
Peters *et al.*, 2000	E. Mass	Arrhythmia symptoms	Yes	No	Yes	NO_2
Fairley, 2003	Santa Clara Co, CA (San Jose)	Mortality	Yes	Yes	Yes for peak O_3	NO_2, O_3, CO
Gold *et al.*, 2003	Boston	HRV	Yes	Yes	Yes for O_3	O_3, NO_2, SO_2
Chock *et al.*, 2000	Pittsburgh	Mortality	No	No	No	Several

(**) For Sheppard (2003), the reanalyzed GAM-based 1-P and 2-P results were both significant. However, the GAM code produces a biased standard error that overstates significance levels, and hence GLM-based results are viewed as more reliable and should be used when available. The GLM-based 1-P result is significant, while the GLM-based 2-P result in this paper is insignificant (albeit borderline), and the relative risk level is reduced. Additionally, all four of the seasonal coefficients for the 2-P GLM models are insignificant.

The above findings represent just one of many ways that the new body of acute effects evidence has been found to vary depending on the particular method of estimation. Other sources of variation in the evidence include the methods for accounting for time and weather considerations. The new studies have demonstrated that concerns with variability of epidemiological estimates of risk, which were expressed but not well explored in 1997, are real. Table 1 at the beginning of my testimony shows that even the "best" $PM_{2.5}$ health effects studies that EPA could select for its risk analysis present a highly uncertain picture of whether $PM_{2.5}$ is playing a causal role for acute effects. Even if there is a causal relationship, which is what the current standard assumed when EPA set it in 1997, there appears to be great difficulty in determining what the size of the effect is. The one trend that is clear is that the size of the $PM_{2.5}$ acute mortality estimates found in the many new studies since 1997 are generally lower than the estimates that were available when the current standards were set.

As I have already noted, EPA has chosen to use an evidence-based approach to set the 24-hour standard. EPA has proposed to tighten the 24-hour standard from the current level of 65 m^3 to 35 m^3. This decision was made even though the quantitative risk estimates based on these studies are lower and statistically weaker than they were when the standard was set. Nevertheless, it is true that there are many more studies available now than at the time of the standard, and it is relevant to ask if this new body of evidence might provide a better indication of a 98th percentile $PM_{2.5}$ level where observed effects start to drop off. EPA has attempted to make such a case for a cut-off point of 35 m^3 in the Proposed Rule.[18] I have gone through that case very carefully, and I have found it incomplete. I will state what I found in general terms here.[19]

EPA's verbal summary of its evidence-based approach used a selected subset of ten $PM_{2.5}$ mortality studies. I found another eight such acute studies of United States or Canadian mortality cited in the Criteria Document, that used actual meas-

[18] See 71 FR 2649.

[19] My written comments to EPA that are being submitted into the record with this testimony provide complete documentation of my review of the literature and application of an evidence-based approach for the 24-hour standard. Although the full discussion includes both mortality and morbidity studies, I only summarize the mortality findings here. However, the patterns I describe are similar in studies of hospitalizations, emergency room visits, and frequency of "symptoms" that are not severe enough to entail a hospital visit.20 Further, although a majority of estimates are statistically significant because the paper focused on the lag period that was found to be significant in the simplest, 1-pollutant starting point of the analysis, the single other alternative 1-pollutant result found a negative risk estimate, implying no risk at all.

urements of $PM_{2.5}$, and that did not appear to have any unreanalyzed statistical problems associated with the GAM software. (If a single paper reports results for more than one city, I treat each city as a separate "study".) Six of the eighteen studies that I considered are the original "Six Cities" used to set the current standard. All of the others are studies published between 1997 and the cut-off time for consideration in this review cycle.

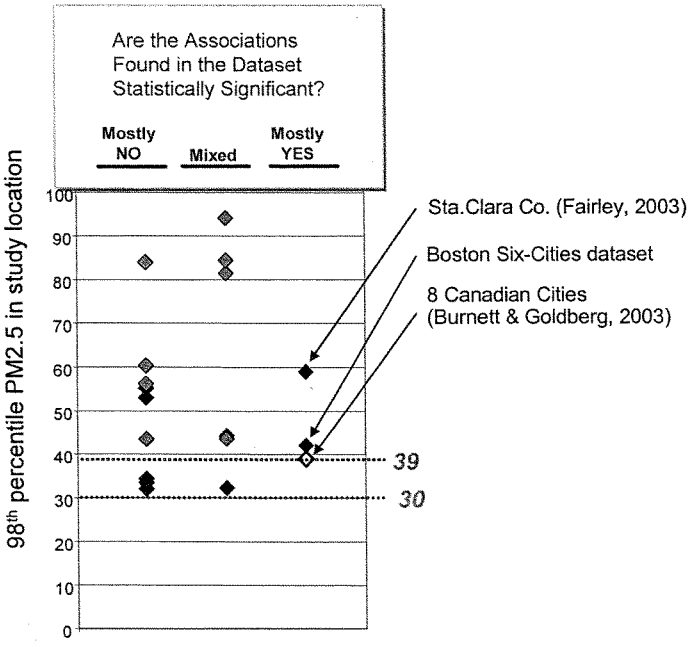
I read each study, and determined whether all the $PM_{2.5}$ estimates reported in a study were "more often insignificant than significant", "a near 50-50 mix", or "more often significant than insignificant." After categorizing them in this way, I found that there is no clear pattern where statistically significant results tend to be found for studies with higher $PM_{2.5}$ levels, and that increasingly mixed evidence is found in studies with progressively lower $PM_{2.5}$ levels. Figure 3 graphically summarizes my findings for the mortality studies. It shows that many of the data sets with the highest 24-hour average $PM_{2.5}$ levels demonstrate the least likelihood of a statistically-significant association with mortality. This is contrary to what EPA states in its discussion of the evidence-based approach in the Proposed Rule. I attribute the difference to the fact EPA considered only a selected set of the new studies, and not the more complete set that I identified. (The ten studies EPA considered are shown as blue diamonds in Figure 3, while the additional eight studies that I also considered are shown as red diamonds in Figure 3.)

Figure 3 also shows that I determined that only 3 of the 18 studies found statistically significant $PM_{2.5}$ effects for a majority of the methods of estimation that they reported. Of these:

- One is for eight Canadian cities combined by Goldberg and Burnett (2003), which I described earlier in this testimony. Its 98th percentile value is about 39 m^3, but this value has the same flaw that I described for its annual average—the actual peak exposures faced by people in the eight separate cities ranged from 27 to 48 m^3, and there is no information to indicate which of the various city-specific 98th percentiles might be accounting for the effects estimated when all cities are combined.
- Another of these was Fairley (2003) for Santa Clara County, CA, with a 98th percentile value of 59 m^3, which I also discussed above. This study had very high exposures at first, and we have no idea whether the statistical significance is related to the earlier high levels, or equally attributable to later, lower $PM_{2.5}$ peaks.[20]
- The third is the Boston data from the Harvard Six Cities study that served as the basis for the current $PM_{2.5}$ NAAQS in 1997. The magnitude and statistical significance of the association observed in this data set has been reduced in reanalyses since 1997, and none of these estimates include consideration of the potential role of any gaseous pollutants in explaining these associations.

[20] Furthery, although a majority of estimates are statistically significant because the paper focused on the lag period that was found to be significant in the simplest, 1-pollutant starting point of the analysis, the single other alternative 1-pollutant result found a negative risk estimate, implying no risk at all.

Figure 3. Summary of the Evidence of Statistically Significant Associations between $PM_{2.5}$ and Acute Mortality in 18 Locations.
(Source: Written comments of Anne E. Smith on EPA's Proposed Rule, April 2006. Copy of these comments is submitted with this testimony)



In conclusion, the evidence regarding a causal relationship between short-term exposures to $PM_{2.5}$ and health has not strengthened since 1997. To draw this conclusion, one must consider more than just the number of new studies that have reported at least one statistically significant association; one must also explore the extent to which the effects reported in these studies remain statistically significant under a range of different plausible methods for making such estimates. In particular, the new evidence strongly suggests that many or most of these associations may actually be attributable to a gaseous pollutant, not $PM_{2.5}$.

But even setting aside the weaknesses in the statistical evidence, EPA's evidence-based approach for where to set a 24-hour standard for $PM_{2.5}$ leads us right back to the very data set on which the current $PM_{2.5}$ NAAQS were based in the Boston "Six Cities" data set. Thus, the evidence-based approach that EPA is trying to apply provides little additional insight beyond the simple point that I started my testimony with: the quantitative estimates of risks remaining at the current standards are lower now than when they were determined to offer an "adequate margin of safety." They therefore do not support a tightening of the current NAAQS.

INTEGRATED ANALYSES OF ALTERNATIVE RESULTS MAY HELP INFORM NAAQS DECISIONS BETTER

It is easy to feel lost regarding how to effectively interpret a plethora of alternative studies, and of alternative risk estimates within each study. EPA's method in performing risk analyses has been to rely on a single estimate that it selects from the large pool of alternatives, and to base its summaries of quantitative risk esti-

mates on that single estimate. In many of these summaries, even the statistical errors associated with that one estimate are often not reported. Some, but not all, of the remaining alternative estimates are studied through "sensitivity analyses." However, these are usually relegated to the back pages of a technical support document. The result of this approach is that the degree of certainty about the risk estimates becomes greatly overstated by the time summary results reach the eyes of decision makers, advisors, and the public. Further, the method of selecting the single risk estimate to rely on for the primary analysis can lead to a substantial bias in the quantitative risk estimates reported.

There are alternative methods for performing risk assessments that integrate multiple alternative risk estimates, and even key uncertainties that remain purely judgmental. These methods are sometimes called probabilistic analysis, or integrated uncertainty analysis. EPA has not used such methods in the documents supporting the Administrator's decision on the $PM_{2.5}$ NAAQS, such as the Staff Paper. I believe that such methods could be very useful, and would reveal better the true extent of uncertainty that I have tried to characterize qualitatively in my testimony above.

In 2003, at an early stage of the drafting of the current risk assessment, I prepared some illustrative examples of an integrated uncertainty analysis to show how the reams and reams of sensitivity results in the risk assessment document could be condensed to more decision-relevant information. The results of that illustrative analysis remain of some interest:

• Using just the alternative long-term exposure studies in the Criteria Document, I found that there could be about a 40 percent probability that there would be no long-term mortality benefit from tightening the current NAAQS. I also estimated that the probability that actual longterm mortality would be less than the primary risk estimate that EPA reports in its risk analysis is about 75 percent.

• I did a less thorough example for the short-term mortality risk, based only on Los Angeles. (Short-term risk estimates and their uncertainty vary by city). For Los Angeles, I estimated a 42 percent probability there would be no benefits from tightening the standard from the current level when using only the risk estimates that EPA had itself cited in its risk analysis, and a 64 percent probability that acute risk reductions would be lower than EPA's primary risk estimate.[21]

These probability estimates were based solely on actual estimates in the new body of literature on $PM_{2.5}$ mortality, and do not include any external judgments such as whether any of these estimates can be interpreted as causal, whether some particles are more toxic than others, or the hypothetical presence of a threshold. (Consideration of these issues would raise the probabilities that I calculated.) They are strictly based in the published evidence reviewed in the Criteria Document. They are thus indicative of the degree of uncertainty that the published studies themselves reveal.

I believe that the process of decision making leading up to the point where a new rule is proposed would benefit greatly if such a synthesis of statistical and modeling uncertainties were to be developed as a part of that process. Controversies would remain regarding the judgments that are necessary for such estimates, but if they are conducted in an open manner, with ample opportunity for public review and comment, more insight about the overall implications of the body of scientific evidence would be created before a decision must be made than we have at the present moment. I emphasize that this should be done during the NAAQS review cycle, with opportunities for public review and comment, before a rule is proposed.

RESPONSES BY ANNE SMITH TO ADDITIONAL QUESTIONS FROM SENATOR VOINOVICH

Question 1. As you stated in your statement, EPA, Dr. Thurston, and others seem to be ignoring your argument that the acceptable level of risk identified in 1997 has gone down. This is a very intuitive and convincing argument. If risk has gone down, then the standard should not be revised. I would like to give you more time to explain this argument and respond to the other side.

Response. The current standards for $PM_{2.5}$ were set by EPA in 1997 with full knowledge that these standards would not reduce health risks to zero. The risk analysis produced by EPA at that time reported positive estimates of risk remaining at the current standard. EPA's risk analysis also explicitly estimated lower levels of risk for alternative, tighter standards than the standard that EPA ultimately se-

[21] For documentation of these calculations, see Smith (2003).

lected. EPA had to defend the risk level that it adopted in court. The DC Circuit Court of Appeals ruled that the risks associated with the current $PM_{2.5}$ standards were "requisite" to protect the public health with an adequate margin of safety, as required by the Clean Air Act.[1] ("Requisite" means that the standards are "not lower or higher than is necessary" to protect public health with an adequate margin of safety.[2]) Thus, the level of risk estimated in 1997 for the current standards was associated with an adequate margin of safety.

As I wrote in my statement: "In thinking about whether to tighten either the annual or daily standard, one might ask, what has changed in our knowledge since 1997 that would undermine the Administrator's 1997 judgment that the current $PM_{2.5}$ NAAQS are neither more nor less stringent than necessary to protect the public health with an adequate margin of safety? A thorough review of the new evidence suggests that the margin of safety that the Administrator selected in 1997 is likely to be larger than was thought at the time."

As I documented in my written testimony to the Committee, EPA's own estimates of health risks remaining at the current standards are lower now than the earlier 1997 estimates. They are lower in all cities for long-term exposure risks, and they are lower for short-term exposure risks in 6 of the 8 cities that are in the current risk analysis.

I presented my comparison of risk estimates from 1997 to those estimated today in person to EPA air office staff on several occasions, and never heard any disagreement regarding its factual accuracy. I have never heard secondhand of EPA staff disagreeing with these facts in any subsequent meetings or conversations where I was not present. I also submitted detailed documentation of the risk comparisons in my written comments to EPA on the draft risk analysis documents and staff paper for $PM_{2.5}$. However, EPA did not provide any such comparative analysis to its Clean Air Scientific Advisory Committee (CASAC) during the course of CASAC's deliberations leading up to CASAC's recommendation to EPA for tightening the $PM_{2.5}$ standards. Thus, CASAC members were probably unaware of these facts when they made their recommendation.[3]

EPA is clearly aware of these facts, however, and even partially acknowledged them at the time that it published the Federal Register notice for the proposed new $PM_{2.5}$ standards:

> With respect to short-term exposure mortality and morbidity ... [c]omparing the risk estimates for the only two specific locations that were included in both the prior and current assessments, the magnitude of the estimates associated with just meeting the current annual standard, in terms of percentage of total incidence, is similar in one of the locations (Philadelphia) and the current estimate is lower in the other location (Los Angeles). . . . With respect to long-term exposure mortality risk estimates, the estimates in terms of percentage of total incidence are very similar for the two specific locations included in both the prior and current assessments.[4]

This quote is only a partial acknowledgment of the risk comparison, however, because it only mentions two cities' risk estimates, and for those two cities, one risk estimate rose and the other fell. However, there were actually 8 cities in the current risk analysis, and the risk estimate has fallen in 6 of those 8. Further, the statement that risk estimates are "similar" for long-term exposure does not reveal that the current risk estimate is actually lower. Notably, this statement was made only after EPA had finalized the Staff Paper, and after CASAC had made its recommendations.

Dr. Thurston, in his testimony before the committee, attempted to rebut my argument in the following way:

> You know, I think that unfortunately what Dr. Smith has done here is exactly what she accuses EPA of having done. She has cherry- picked certain results that support her position and unfortunately hasn't looked in a balanced unbiased way at this question.[5]

This is a completely false statement, as anyone should be able to tell from the record of the hearing: my comparison is based only on EPA's own numbers. All that

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[1] United States Court of Appeals for the District Of Columbia Circuit in American Trucking Associations, Inc., et al., v. Environmental Protection Agency, No. 97-1440, March 26, 2001.

[2] Ibid, p. 10.

[3] Although my risk comparisons are based on material that EPA placed into the record during both the 1997 and current rulemakings, CASAC could not be expected to have easily made such comparisons on their own. For one, the relevant measures of risk are not readily observed given the formats in which EPA has presented its risk findings. Further, they require one to consult risk analysis documents from 10 years ago.

[4] PR, p. 2640.

[5] EPW Committee Hearing transcript, July 19, 2006.

I have done is assemble the information from EPA documents and repeat it in a format that allows a direct comparison. I made this clear in my own oral statement:

Risks due to long-term mortality have fallen in every location. The risks due to daily exposure, which vary by city, have fallen in six of the eight cities that are in EPA's risk analysis. This new information suggests that the margin of safety provided by the current standards is actually greater than we originally thought. What I have told you so far is based entirely on EPA's own point estimates of risk.[6]

Dr. Thurston's comment is an attempt to sidestep the inconvenient truth that even EPA's analyses support the conclusion that the estimates of mortality risk associated with the current ambient $PM_{2.5}$ standard have fallen since the time that they were deemed acceptable by EPA and the Court. His sidestepping tactic may be useful in a purely oral exchange, but the transcript makes it clear that his comment has no substance or merit in refuting my argument.

I did go on to describe how the evidence on lower risk estimates is even stronger when one looks at the full body of evidence in the studies that have been released since 1997, rather than just using EPA's own numbers. However, it is illogical to claim that I have "cherry-picked" the evidence when I show that the downward risk trends would be more pronounced if I use almost any other risk estimate than those used by EPA. This is the opposite of cherry-picking.

I would also like to directly address Dr. Thurston's own argument that there is new evidence to support tightening the annual standard. The essence of his argument is that further analyses of the American Cancer Society study find that there is no evidence of a threshold in the long-term exposure risk data below the level of the current annual standard. He presents a figure (Figure 3 in his written testimony), that indicates that the risks from $PM_{2.5}$ exposure extend below 15 m^3, supporting a reduction in the annual $PM_{2.5}$ standard at this time.[7] While it is true that this specific figure had not been produced at the time that the current annual standard was established, it does not provide a case for tightening the standard to a level below 15 m^3. Dr. Thurston's implicit premise is that the $PM_{2.5}$ standard should be a zero-risk standard, which was not and still is not the premise of EPA (and upheld by the Court). Moreover, EPA assumed that a causal relationship between $PM_{2.5}$ and long-term mortality risk did exist down to 9 m^3

in its risk analyses when it chose to set the annual standard at 15 m^3. Thus, the "new" information that Dr. Thurston says supports reducing the standard to a lower level was already assumed to exist in the 1997 risk analyses.

Question 2. Your testimony indicates that EPA has selectively used study results in developing the proposal. EPA emphasized studies that show associations between particulates and negative health effects and ignored those that do not. You also point out that in many of the studies relied upon, the associations become statistically insignificant when another pollutant is included in the analysis. Please elaborate.

Response. Each of the epidemiological studies of $PM_{2.5}$ risk contains many different alternative estimates of risk. Different estimates of risk are possible because the researchers have to specify what set of risk factors in addition to $PM_{2.5}$ will be simultaneously accounted for when estimating the quantitative amount of risk that is associated with $PM_{2.5}$. For example, one can estimate a $PM_{2.5}$ risk level with or without also including the humidity level. $PM_{2.5}$ concentrations may tend to be higher on days of higher humidity, yet humidity is known to be a physiological stressor and there is an established increased risk of a sick individual dying on the relatively more humid days of a year. If humidity is left out of the analysis, then $PM_{2.5}$ could serve as a "proxy" for the missing humidity information, and this could bias the level of risk estimated for $PM_{2.5}$ upwards. All of the current studies take care to address humidity, because its role as a stressor is so well established. However, there is no objective method for determining exactly how to specify humidity. Further, there are many alternative ways to specify a wide range of other factors that also need to be accounted for to avoid bias in the $PM_{2.5}$ effect, and not all of the relevant health stressors are known. Thus, researchers tend to try different methods for incorporating non-$PM_{2.5}$ determinants of health, and each different method results in a different $PM_{2.5}$ risk estimate.

Sometimes two seemingly comparable methods produce dramatically different results. For example, the study of $PM_{2.5}$ risk in Santa Clara County, CA by Fairley (2003) reports results of two estimates that differ in one single way: in one case,

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[6] Ibid, emphasis added.

[7] Statement of Dr. George D. Thurston, Sc. D. to the Committee on Environment and Public Works of the United States Senate RE: the Science and Risk Assessment of Particulate Matter (PM) Air Pollution Health Effects, July 19, 2006.

mortality risk one a particular day was compared to $PM_{2.5}$ levels on the same day, and in the second, mortality risk on each day was compared to $PM_{2.5}$ levels that occurred the day before.[8] The first of these finds a positive and statistically significant association between rises in $PM_{2.5}$ and mortality risk. The second finds the opposite: a negative association (i.e., mortality risk tended to be lower if $PM_{2.5}$ on the day before had been relatively high). There is no objective statistical means for determining whether one of these statistical findings is closer to the truth than the other, but they produce extremely different estimates of health risk from ambient $PM_{2.5}$. In this case, EPA has selected the positive and statistically significant result for use in its risk analysis, and EPA makes no mention of the opposite alternative result that it could have selected from the same paper.

One important class of researcher judgments where very different risk estimates can result is that related to "single-pollutant" versus "multi-pollutant" estimation methods. Most commonly, the only pollutant that is included in the statistical estimation procedure is $PM_{2.5}$ (at least, in studies that report $PM_{2.5}$ risk estimates). If any other pollutant has an association with mortality risks, then $PM_{2.5}$ will be the only pollutant that could adopt its explanatory power and it will do so if $PM_{2.5}$ is statistically correlated with the missing pollutant. As a result, the $PM_{2.5}$ risk estimate will be biased, and may appear falsely to be statistically significant. In a multi-pollutant statistical estimation procedure, $PM_{2.5}$ and one or more other pollutants are all included in the analysis at the same time. Then, each can account for its own role as a health stressor, and the resulting estimates for each pollutant, including $PM_{2.5}$, are less likely to be biased.

I attempted to identify all of the $PM_{2.5}$ health effects studies cited in the Criteria Document (including both mortality and morbidity effects studies) that had reported results of any estimates for $PM_{2.5}$ using a multi-pollutant method of estimation for at least one gaseous pollutant. I found ten such papers among all the new studies that did report a statistically significant association for $PM_{2.5}$. Of these 10, 8 found that $PM_{2.5}$ lost its ability to explain mortality risk when studied using a 2-pollutant method. (In the other two studies, both the $PM_{2.5}$ and gaseous pollutant retained statistical explanatory power.) This suggests a reasonable concern that $PM_{2.5}$ may not be the pollutant that is actually causing the reported health risks. However, EPA's risk analysis is relying solely on the one-pollutant results from each of these papers, and thus is overstating the degree of confidence in these results. Notably, 2 of the 8 cities in EPA's risk analysis are being estimated using single-pollutant estimates from papers that also reported that $PM_{2.5}$ risk estimates are not statistically significant if estimated in a two-pollutant model: Los Angeles and Philadelphia.[9], [10] In both cases, the authors concluded that a different pollutant had more explanatory power than $PM_{2.5}$, yet EPA has used only the obviously biased estimate of $PM_{2.5}$ risk that came from the single-pollutant methods that the original researchers have repudiated. (Even with use of the one-pollutant estimate, EPA's mortality risk estimate for Los Angeles is not statistically significant.)

In my response so far, I have explained how EPA has been selective in the way it has made risk estimates for 3 of the 8 cities in its mortality risk assessment (i.e., for Santa Clara County/San Jose, Philadelphia, and Los Angeles). Problems with the remaining 5 cities risk estimates also deserve mention:

- For both Pittsburgh and Detroit, the risk estimate that EPA uses is not statistically significant.[11], [12]
- For Phoenix, there are 2 other papers by other authors that use the same $PM_{2.5}$ data set.[13] One of the other papers finds no $PM_{2.5}$ association at all (but does find

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[8] Fairley, D. (2003) Mortality and Air Pollution for Santa Clara County, CA, 1989-1996 Revised Analyses of Time-series Studies of Air Pollution and Health, Special Report. Boston, MA: Health Effects Institute; pp. 97-106.

[9] Moolgavkar, S. H. (2003) "Air Pollution and Daily Deaths and Hospital Admissions in Los Angeles and Cook Counties" Revised Analyses of Time-series Studies of Air Pollution and Health, Special Report Boston, MA: Health Effects Institute; pp. 183-198.

[10] Lipfert, F. W.; Morris, S. C.; Wyzga, R. E. (2000a) Daily Mortality in the Philadelphia Metropolitan Area and Size-classified Particulate Matter Journal of the Air & Waste Management Association 15011513.

[11] Chock, D. P.; Winkler, S.; Chen, C. (2000) A Study of the Association Between Daily Mortality and Ambient Air Pollutant Concentrations in Pittsburgh, Pennsylvania Journal of the Air & Waste Management Association 50: 1481-1500.

[12] Ito, K. (2003) Associations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit, Michigan. Revised Analyses of Time-series Studies of Air Pollution and Health. Special report. Boston, MA: Health Effects Institute; pp. 143-156.

[13] The paper EPA relies on is: Mar, T. F.; Norris, G. A.; Larson, T. V.; Wilson, W. E.; Koenig, J. Q. (2003) "Air Pollution and Cardiovascular Mortality in Phoenix, 1995-1997" Revised Analyses of Time-series Studies of Air Pollution and Health, Special Report Boston, MA: Health Effects Institute; pp. 177-182.

a coarse fraction association).[14] The third paper reports a $PM_{2.5}$ association only above a threshold. EPA's risk assessment does not include such a threshold.[15]

• Risk estimates for both St. Louis and Boston are based on single-pollutant methods because the researchers do not report any multi-pollutant methods at all.[16], [17] However, for both cities, the authors do report a number of different $PM_{2.5}$ risk estimates using different ways of accounting for non-pollutant variables that also account for changes in mortality risk and EPA has adopted the estimate for each city that produces the highest level of $PM_{2.5}$ risk. Several of the alternative estimates are also not statistically significant (particularly for St. Louis). There is no reason to expect those higher risk estimates to be more valid than the alternative estimates reported by the researchers. In summary, there is a substantial amount of uncertainty associated with risk estimates for all eight of the cities in EPA's current risk assessment. The way EPA has selected a single risk estimate out of the many estimates provided in the original epidemiological studies for each city has a clear tendency to overstate the level of $PM_{2.5}$ mortality risk and to understate the uncertainties.

Question 3. Please summarize the concerns that you have with the three studies that CASAC relied on in recommending a tightened annual standard.

Response. CASAC notes that there are three new acute studies that find $PM_{2.5}$ associations with mortality at annual averages below the current annual standard (all with reported annual averages in the range of 13 to 14 g/m^3). Most importantly, there are logical flaws in setting an annual standard based on studies of daily mortality risk. Acute risks are associated with increases in $PM_{2.5}$ on certain days and there is no logical link between the the annual average conditions at a given location and the riskiness of spikes in $PM_{2.5}$ at that location.

However, even if one were to want to set an annual standard based on short-term exposure risk studies, each of these papers presents reasons to be concerned about the prospect of using them as the basis for a national standard:

Goldberg and Burnett (2003).[18] This study reports a $PM_{2.5}$ association for eight Canadian cities combined. The annual average of 13.3 m^3 is an average over all of the eight cities, while the annual averages in the individual cities vary from 9.5 m^3 to 17.7 m^3. There are no city-specific results reported to help indicate whether the estimate of an acute effect is due to effects in each of the eight cities, or only in a few.[19] Evidence in the paper suggests that there may in fact be different effects in each city.

Another concern with this study is that it is a reanalysis of a more comprehensive study that included consideration of the role of gaseous pollutants as well.[20] The original study concluded that the gaseous pollutants had a much greater ability to explain mortality risks than both $PM_{2.5}$ and $PM_{10\text{-}2.5}$ combined. However, when the paper had to be reanalyzed, the authors did not reanalyze the portions that considered gaseous pollutants in conjunction with particulate pollution, and so this finding is no longer discussed.

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[14] Clyde, M. A.; Guttorp, P.; Sullivan, E. (2000) "Effects of Ambient Fine and Coarse Particles on Mortality in Phoenix, Arizona" Seattle, WA: University of Washington, National Research Center for Statistics and the Environment; NRCSE technical report series, NRCSE-TRS no. 040.

[15] Smith, R. L.; Spitzner, D.; Kim, Y.; Fuentes, M. (2000) "Threshold Dependence of Mortality Effects for Fine and Coarse Particles in Phoenix, Arizona" Journal of the Air & Waste Management Association 50: 1367-1379.

[16] Klemm, R. J.; Mason, R. (2003) "Replication of Reanalysis of Harvard Six-City Mortality Study." Revised Analyses of Time-series Studies of Air Pollution and Health. Special report. Boston, MA: Health Effects Institute; pp. 165-172.

[17] Schwartz, J. (2003) "Daily Deaths Associated with Air Pollution in Six US Cities and Short-term Mortality Displacement in Boston" Revised Analyses of Time-series Studies of Air Pollution and Health. Special report. Boston, MA: Health Effects Institute; pp. 219-226.

[18] Burnett, R. T.; Goldberg, M. S. (2003) "Size-fractionated Particulate Mass and Daily Mortality in Eight Canadian Cities." Revised Analyses of Time-series Studies of Air Pollution and Health. Special report. Boston, MA: Health Effects Institute; pp. 85-90.

[19] The other multi-city $PM_{.5}$ mortality studies (based on the Six Cities data set) report effects by individual city as well as for the combined set. This was the data on which EPA set the current standards, and in doing so, EPA used annual averages for only the individual cities that did have significant effects within the set of six. The lowest such city was Boston, with an annual average of 15.6 um^3, which was the basis for the current annual average standard of 15 m^3.

[20] Burnett, R. T.; Brook, J.; Dann, T.; Delocla, C.; Philips, O.; Cakmak, S.; Vincent, R.; Goldberg, M. S.; Krewski, D. (2000) "Association Between Particulate- and Gas-phase Components of Urban Air Pollution and Daily Mortality in Eight Canadian Cities" In: Grant, L. D., ed. PM2000: Particulate Matter and Health. Inhalation Toxicology 12(suppl. 4): 15-39.

• Fairley (2003).[21]This study used data from Santa Clara County, CA, over a seven year period, and during that time pollution levels were falling dramatically. Although the annual average $PM_{2.5}$ that is attributed to this study is 13.6 m^3, the annual average was as high as 18.4 m^3 at the start, and fell progressively to 9.5 m^3 by the end of the 7 years studied.[22] Peak levels of $PM_{2.5}$ were also falling, starting at a 98th percentile of 88 m^3 for the first year and ending at 25 m^3. Such a wide range of $PM_{2.5}$ levels within this one city's data set begs the question: Are the reported acute effects relationship driven largely by the high levels in the early years, or are they also evident in the later years? This highly relevant question is never mentioned, let alone analyzed, by the authors. Lacking any exploration of such an obviously relevant issue, it would seem a dubious proposition to use the annual average over the entire time period in this one study as the basis for a national ambient standard.

Another concern with this study is that it reports $PM_{2.5}$ risk estimates for two alternative methods of estimation, both of which are reasonable. One method considers whether deaths tend to fluctuate with the same day's $PM_{2.5}$ levels and the other method considers whether deaths tend to fluctuate with the previous day's $PM_{2.5}$ levels. The same-day estimate finds the positive association that this study is known for, but the estimate based on $PM_{2.5}$ on just the previous day is actually in the negative direction. Complete reversal of evidence of a $PM_{2.5}$ mortality effect by considering $PM_{2.5}$ levels only 24 hours apart in time presents a concern for interpreting the study's same-day estimate as a causal one. However, there is no discussion of what these conflicting results might mean.

• Mar et al. (2003).[23] This study considered acute risks in Phoenix, AZ, with annual average $PM_{2.5}$ levels of 13.5 m^3. There are ten estimates of $PM_{2.5}$ risk in the paper, and only three of them are significant. More importantly, this is not the only paper that studied the ability of this same set of $PM_{.5}$ data to explain acute mortality risks in Phoenix. One of the other studies found that $PM_{2.5}$ did not have any explanatory power, and found instead that the coarse fraction of PM had explanatory power.[24] The third study found evidence that there is a threshold below which $PM_{2.5}$'s apparent ability to explain changes in daily mortality disappeared.[25] That threshold appeared to be above 20 m^3. If there is a threshold, then the rationale for a linkage between annual average $PM_{2.5}$ and acute risks simply falls apart.

A final concern with all three of the Phoenix studies is that none of them considered whether the $PM_{2.5}$ effect would remain if pollutants such as CO, SO_2, ozone, or NO_2 were also included in the analysis. This is a critical gap in many of the current studies because the new body of papers on $PM_{2.5}$ health effects reveals that $PM_{2.5}$ effects usually disappear when one of the gaseous pollutants is explored. (My response to Question 2 above addresses this point in more detail.)

STATEMENT OF JOHN STEPHENSON, DIRECTOR, NATURAL RESOURCES AND ENVIRONMENT, U.S. GENERAL ACCOUNTABILITY OFFICE

Mr. Chairman and members of the committee, I am pleased to be here today as the committee considers the science and risk assessment supporting the Environmental Protection Agency's (EPA) proposed revisions to the national air quality standards for particulate matter. A large body of scientific evidence links exposure to particulate matter a ubiquitous form of air pollution commonly referred to as soot to serious health problems, including asthma, chronic bronchitis, heart attack, and premature death. Under the Clean Air Act, EPA periodically reviews the appropriate air quality level at which to set national standards to protect the public against the health effects of particulate matter. As you are aware, EPA proposed revisions to the particulate matter standards in January 2006 and issued a draft regulatory impact analysis of the revisions expected costs and benefits.

EPA's estimates of the expected benefits from its air pollution regulations have often been controversial, and the methods the agency has used to prepare these estimates have been questioned. In 2000, at the direction of the Senate Appropriations Committee, EPA asked the National Academies (Academies) to evaluate EPA's overall methodology for estimating the health benefits of proposed air regulations. In 2002, the Academies issued a report that made recommendations focusing on con

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[21] Fairley, op. cit.

[22] To know this, one must go back and read the original study that Fairley (2003) reanalyzes: Fairley, D. (1999) Daily Mortality and Air Pollution in Santa Clara County, CA: 1989-1996? Environmental Health Perspectives 107: 637-641.

[23] Mar et al., op. cit.

[24] Clyde et al., op. cit.

[25] Smith et al., op. cit.

ducting more rigorous assessments of uncertainty, increasing the transparency of how EPA estimates benefits, conducting more detailed analyses of exposure, and estimating the benefits of each regulatory option under consideration. My testimony summarizes the highlights of our report being released today on the extent to which EPA applied the recommendations made by the Academies to its January 2006 proposed revisions to the particulate matter standards.[1] Our report provides a more detailed discussion of each recommendation, including whether and how EPA applied it to the regulatory impact analysis on particulate matter.

SUMMARY

While the National Academies report generally supported EPA's overall approach to estimating benefits, it included 34 recommendations for improvements. EPA has begun to change the way it conducts and presents its analyses of health benefits in response to the National Academies? recommendations. In the case of the January 2006 proposed rule on particulate matter standards, EPA applied, at least in part, about two-thirds of the recommendations to its particulate matter health benefit analysis; it applied 8 and partially applied 14 more. For example, in applying the recommendations, EPA evaluated how benefits might change given alternative assumptions and discussed sources of uncertainty not included in the benefit estimates. In addition, EPA applied an alternative technique for evaluating one important source of uncertainty in its analysis the uncertainty underlying the causal link between exposure to particulate matter and premature death. Consistent with the National Academies recommendation to assess uncertainty by developing ranges of estimates of benefits and specifying the likelihood of attaining those levels of benefits, EPA systematically gathered expert opinions about this link and developed ranges reflecting the experts confidence in attaining reductions in premature death expected from the proposed revisions. However, the health benefit analysis did not assess how the benefit estimates would vary in light of other key uncertainties as the Academies recommended. Consequently, EPA's response represents a partial application of the recommendation. Agency officials told us that ongoing research and development efforts will allow EPA to gradually make more progress in applying this and other recommendations to future analyses.

EPA did not apply the remaining 12 recommendations to the analysis, such as the recommendation to evaluate the impact of using the assumption that the components of particulate matter are equally toxic. EPA officials viewed most of these 12 recommendations as relevant to its health benefit analyses but noted that the agency was not ready to apply specific recommendations because of, among other things, the need to overcome technical challenges stemming from limitations in the state of available science. For example, EPA did not believe that the state of scientific knowledge on the relative toxicity of particulate matter components was sufficiently developed to include it in the January 2006 regulatory impact analysis, but the agency is sponsoring research on this issue.

BACKGROUND

EPA is required by the Clean Air Act to conduct reviews of the National Ambient Air Quality Standards (NAAQS) for the six criteria pollutants, including particulate matter, every 5 years to determine whether the current standards are sufficient to protect public health, with an adequate margin of safety. If EPA decides to revise the NAAQS, the agency proposes changes to the standards and estimates the costs and benefits expected from the revisions in an assessment called a regulatory impact analysis. In January 2006, EPA prepared a regulatory impact analysis for one such rule?particulate matter that presented limited estimates of the costs and benefits expected to result from the proposed particulate matter rule. EPA developed the estimates by, for example, quantifying the changes in the number of deaths and illnesses in five urban areas that are likely to result from the proposed rule.

The National Academies' 2002 report examined how EPA estimates the health benefits of its proposed air regulations and emphasized the need for EPA to account for uncertainties and maintain transparency in the course of conducting benefit analyses. Identifying and accounting for uncertainties in these analyses can help decision makers evaluate the likelihood that certain regulatory decisions will achieve the estimated benefits. Transparency is important because it enables the public and relevant decision makers to see clearly how EPA arrived at its estimates and conclusions. Many of the recommendations include qualifying language indicating that it

[1] See GAO, Particulate Matter: EPA Has Started to Address the National Academies Recommendations on EstimatingHealth Benefits, but MoreProgress Is Needed, GAO-06-780 (Washington, DC: July 14, 2006).

is reasonable to expect that they can be applied in stages, over time; moreover, a number of the recommendations are interrelated and, in some cases, overlapping. Soon after the National Academies issued its report, EPA roughly approximated the time and resource requirements to respond to the recommendations, identifying those the agency could address within 2 or 3 years and those that would take longer. According to EPA officials, the agency focused primarily on the numerous recommendations related to analyzing uncertainty. As is discussed below, EPA applied some of these recommendations to the particulate matter analysis.

EPA Applied Some, but Not All, of the National Academies Recommendations to the Particulate Matter Regulatory Impact Analysis

EPA applied either wholly or in part approximately two-thirds of the Academies recommendations in preparing its January 2006 particulate matter regulatory impact analysis and continues to address the recommendations through ongoing research and development. According to EPA, the agency intends to address some of the remaining recommendations in the final rule and has undertaken research and development to address others.

Recommendations EPA Applied or Partially Applied to Its Particulate Matter Health Benefit Analysis

The January 2006 regulatory impact analysis on particulate matter represents a snapshot of an ongoing EPA effort to respond to the National Academies recommendations on developing estimates of health benefits for air pollution regulations. Specifically, the agency applied, at least in part, approximately two-thirds of the recommendations 8 were applied and 14 were partially applied by taking steps toward conducting a more rigorous assessment of uncertainty by, for example, evaluating the different assumptions about the link between human exposure to particulate matter and health effects and discussing sources of uncertainty not included in the benefit estimates. According to EPA officials, the agency focused much of its time and resources on the recommendations related to uncertainty. In particular, one overarching recommendation suggests that EPA take steps toward conducting a formal, comprehensive uncertainty analysis the systematic application of mathematical techniques, such as Monte Carlo simulation and include the uncertainty analysis in the regulatory impact analysis to provide a more realistic depiction of the overall uncertainty in EPA's estimates of the benefits.[2]

Overall, the uncertainty recommendations call for EPA to determine (1) which sources of uncertainties have the greatest effect on benefit estimates and (2) the degree to which the uncertainties affect the estimates by specifying a range of estimates and the likelihood of attaining them. In response, EPA examined a key source of uncertainty its assumption about the causal link between exposure to particulate matter and premature death and presented a range of expected reductions in death rates. EPA based these ranges on expert opinion systematically gathered in a multiphased pilot project. The agency did not, however, incorporate these ranges into its benefit estimates as the National Academies had recommended.

Moreover, the Academies recommended that EPA's benefit analysis reflect how the benefit estimates would vary in light of multiple uncertainties. In addition to the uncertainty underlying the causal link between exposure and premature death, other key uncertainties can influence the estimates. For example, there is uncertainty about the effects of the age and health status of people exposed to particulate matter, the varying composition of particulate matter, and the measurements of actual exposure to particulate matter. EPA's health benefit analysis, however, does not account for these key uncertainties by specifying a range of estimates and the likelihood of attaining them. For these reasons, EPA's responses reflect a partial application of the Academies recommendation.

In addition, the Academies recommended that EPA both continue to conduct sensitivity analyses on sources of uncertainty and expand these analyses. In the particulate matter regulatory impact analysis, EPA included a new sensitivity analysis regarding assumptions about thresholds, or levels below which those exposed to particulate matter are not at risk of experiencing harmful effects. EPA has assumed no threshold level exists that is, any exposure poses potential health risks.[3] Some

[2] Monte Carlo simulation refers to a computer-based analysis that uses probability distributions for key variables, selects random values from each of the distributions simultaneously, and repeats the random selection over and over. Rather than presenting a single outcome such as the mostly likely or average scenario Monte Carlo simulations produce a distribution of outcomes that reflect the probability distributions of modeled uncertain variables.

[3] Recent EPA analyses used the natural background concentrations of particulate matter, rather than zero, for its assumption of no threshold level. The National Academies supported the assumption of no threshold level, but it recommended that EPA conduct a consistent and transparent sensitivity analysis to consider various threshold levels.

experts have suggested that different thresholds may exist, and the National Academies recommended that EPA determine how changing its assumption that no threshold exists would influence the estimates. The sensitivity analysis EPA provided in the regulatory impact analysis examined how its estimates of expected health benefits would change assuming varying thresholds.

In response to another recommendation by the National Academies, EPA identified some of the sources of uncertainty that are not reflected in its benefit estimates. For example, EPA's regulatory impact analysis disclosed that its benefit estimates do not reflect the uncertainty associated with future year projections of particulate matter emissions. EPA presented a qualitative description about emissions uncertainty, elaborating on technical reasons such as the limited information about the effectiveness of particulate matter control programs why the analysis likely underestimates future emissions levels.

Recommendations EPA Did Not Apply to the Particulate Matter Analysis

EPA did not apply the remaining 12 recommendations to the analysis for various reasons. Agency officials viewed most of these recommendations as relevant to its health benefit analyses and, citing the need for additional research and development, emphasized the Agency's commitment to continue to respond to the recommendations. EPA has undertaken research and development to respond to some of these recommendations but, according to agency officials, did not apply them to the analysis because the agency had not made sufficient progress.

For example, EPA is in the process of responding to a recommendation involving the relative toxicity[4] of components of particulate matter, an emerging area of research that has the potential to influence EPA's regulatory decisions in the future.[5] Hypothetically, the agency could refine national air quality standards to address the potentially varying health consequences associated with different components of particulate matter. The National Academies recommended that EPA strengthen its benefit analyses by evaluating a range of alternative assumptions regarding relative toxicity and incorporate these assumptions into sensitivity or uncertainty analyses as more data become available.[6] EPA did not believe the state of scientific knowledge on relative toxicity was sufficiently developed at the time it prepared the draft regulatory impact analysis to include this kind of analysis. In a separate report issued in 2004, the National Academies noted that technical challenges have impeded research progress on relative toxicity but nonetheless identified this issue as a priority research topic. The Clean Air Scientific Advisory Committee also noted the need for more research and concluded in 2005 that not enough data are available to base the particulate matter standards on composition. The Office of Management and Budget, however, encouraged EPA in 2006 to conduct a sensitivity analysis on relative toxicity and referred the agency to a sensitivity analysis on relative toxicity funded by the European Commission.

We found that EPA is sponsoring research on the relative toxicity of particulate matter components. For example, EPA is supporting long-term research on this issue through its intramural research program and is also funding research through its five Particulate Matter Research Centers and the Health Effects Institute. In addition, an EPA contractor has begun to investigate methods for conducting a formal analysis that would consider sources of uncertainty, including relative toxicity. To date, the contractor has created a model to assess whether and how much these sources of uncertainty may affect benefit estimates in one urban area. Agency officials told us, however, that this work was not sufficiently developed to include in the final particulate matter analysis, which it says will present benefits on a national scale.

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[4] Particulate matter is a highly complex mixture comprising particles emitted directly from sources and particles formed through atmospheric chemical reactions. Particles span many sizes and shapes and consist of hundreds of different chemicals. EPA identifies the major components of fine particulate matter as carbon, sulfate and nitrate compounds, and crustal/metallic materials such as soil and ash.

[5] Relative toxicity refers to the premise that different components of particulate matter have different levels of potency affecting premature mortality and illness. In the draft particulate matter regulatory impact analysis, EPA assumed equivalent toxicity, stating that while it is reasonable to expect that the potency of components may vary across the numerous effect categories associated with particulate matter, EPA's interpretation of scientific information considered to date is that such information does not yet provide a basis for quantification beyond using fine particle mass. EPA, Draft Regulatory Impact Analysis for the $PM_{PM-2.5}$ National Ambient Air Quality Standards (Washington, DC, 2006), 3-21.

[6] In the context of the National Academies recommendations, a sensitivity analysis would assess how changes in one or more variables affect the outcome, whereas a comprehensive or formal uncertainty analysis evaluates the probability distributions of multiple variables.

Another recommendation that EPA did not apply to the particulate matter analysis focused on assessing the uncertainty of particulate matter emissions. The National Academies recommended that EPA conduct a formal analysis to characterize the uncertainty of its emissions estimates, which serve as the basis for its benefit estimates.[7] While the agency is investigating ways to assess or characterize this uncertainty, EPA did not conduct a formal uncertainty analysis for particulate matter emissions for the draft regulatory impact analysis because of data limitations. These limitations stem largely from the source of emissions data, the National Emissions Inventory,[8] an amalgamation of data from a variety of entities, including state and local air agencies, tribes, and industry. According to EPA, these entities use different methods to collect data, which have different implications for how to characterize the uncertainty. EPA officials stated that the agency needs much more time to address this data limitation and to resolve other technical challenges of such an analysis. While the final particulate matter analysis will not include a formal assessment of uncertainty about emissions levels, EPA officials noted that the final analysis will demonstrate steps toward this recommendation by presenting emissions data according to the level emitted by the different kinds of sources, such as utilities, cars, and trucks.

Finally, EPA did not apply a recommendation concerning the transparency of its benefit estimation process to the particulate matter analysis. Specifically, the National Academies recommended that EPA clearly summarize the key elements of the benefit analysis in an executive summary that includes a table that lists and briefly describes the regulatory options for which EPA estimated the benefits, the assumptions that had a substantial impact on the benefit estimates, and the health benefits evaluated. EPA did not, however, present a summary table as called for by the recommendation or summarize the benefits in the executive summary. EPA stated in the regulatory impact analysis that the agency decided not to present the benefit estimates in the executive summary because they were too uncertain. Agency officials told us that the agency could not resolve some significant data limitations before issuing the draft regulatory impact analysis in January 2006 but that EPA has resolved some of these data challenges. For example, EPA officials said they have obtained more robust data on anticipated strategies for reducing emissions, which will affect the estimates of benefits. The officials also said that EPA intends to include in the executive summary of the regulatory impact analysis supporting the final rule a summary table that describes key analytical information.

CONCLUDING OBSERVATIONS

While EPA officials said that the final regulatory impact analysis on particulate matter will reflect further responsiveness to the Academies recommendations, continued commitment and dedication of resources will be needed if EPA is to fully implement the improvements recommended by the National Academies. In particular, the agency will need to ensure that it allocates resources to needed research on emerging issues, such as the relative toxicity of particulate matter components, and to assessing which sources of uncertainty have the greatest influence on benefit estimates. The uncertainty of the agency's estimates of health benefits in the draft regulatory impact analysis for particulate matter underscores the importance of uncertainty analysis that can enable decision makers and the public to better evaluate the basis for EPA's air regulations. While EPA officials said they expect to reduce the uncertainties associated with the health benefit estimates in the final particulate matter analysis, a robust uncertainty analysis of the remaining uncertainties will nonetheless be important for decision makers and the public to understand the likelihood of attaining the estimated health benefits.

Mr. Chairman, this concludes my prepared statement. I would be happy to respond to any questions that you or members of the committee may have.

RESPONSES BY JOHN STEPHENSON TO ADDITIONAL QUESTIONS FROM SENATOR VOINOVICH.

Question 1. Mr. Stephenson, your testimony states "The National Academies" 2002 report examined how EPA estimates the health benefits of its proposed air regulations and emphasized the need for EPA to account for uncertainties and main-

[7] Because the precise levels of total emissions are not knowable but rather are approximations based on a sample of measurements, there is uncertainty about the true quantity of emissions.

[8] EPA compiles the National Emissions Inventory, a national database of air emissions data that includes estimates of annual emissions, by source, of air pollutants in each area of the country on an annual basis.

tain transparency in the course of conducting benefit analyses. I agree that transparency is very important. However, right now, EPA is simply telling us that we need to wait until the final rule to see a more complete RIA, how they addressed more of these recommendations, and the inclusion of new science. In your opinion, has EPA "maintained transparency" through this process with the PM standards?

Response. EPA has maintained transparency to the extent feasible under federal rulemaking procedures, which include collecting and reviewing public comments on the proposed rule and submitting the final rule package to the White House Office of Management and Budget for review at least 45 days before the EPA Administrator signs the rule. Proposed and final rules can differ substantially for a number of reasons, including changes made in response to public comments on the proposed rule and the availability of new or more complete data. Therefore, in developing and supporting both proposed and final rules, it is important for agencies to be transparent that is, to provide decision makers and the public with clear and relevant information about the data, assumptions, methodologies, uncertainties, etc., underlying its regulatory impact analyses. As you are aware, the Congressional Review Act of 1996 established an expedited process under which Congress may disapprove a broad range of regulatory rules issued by Federal agencies by enacting a joint resolution of disapproval within 60 days after receiving the rule. The rule would not go into effect without subsequent statutory authorization.

To date we have not identified major shortcomings concerning the transparency of EPA's current rulemaking process for particulate matter standards. For example, in the January 2006 regulatory impact analysis for proposed rule, EPA highlighted the fact that the benefits estimates were based only on five cities and were highly uncertain. According to EPA officials, the agency could not resolve data limitations in time to meet the court-issued deadline for the proposed changes. As you know, EPA plans to provide national estimates of the health benefits in the final rule using more robust data that it has subsequently been able to obtain. Thus, the regulatory impact analyses supporting the proposed and final rules are expected to be significantly different. EPA can maintain transparency in this rulemaking process by clearly discussing in the final regulatory impact analysis due in September 2006 the changes in its approach to estimating the benefits and the Agency's rationale for doing so and by clearly identifying key data, assumptions, methodologies, and uncertainties.

In terms of transparency and the science supporting the rulemaking efforts, EPA made a commitment in the proposed rule to conduct a review and assessment of studies published after the completion of the scientific review of health effects linked to particulate matter emissions. EPA has recently made information about its review of new science available to the public; see EPA, Provisional Assessment of Recent Studies on Health Effects of Particulate Matter Exposure (Research Triangle Park, N.C., 2006). The July 2006 provisional assessment presents EPA's approach to reviewing new studies and summarizes the findings of these studies, including those that evaluated the links between particulate matter components and adverse health effects.

Question 2. Mr. Stephenson, please elaborate on this very important statement that you make at the end of your testimony (quote): ″While EPA officials said they expect to reduce the uncertainties associated with the health benefit estimates in the final particulate matter analysis, a robust uncertainty analysis of the remaining uncertainties will nonetheless be important for decision makers and the public to understand the likelihood of attaining the estimated health benefits."

Response. In estimating potential health benefits stemming from regulatory actions, some level of uncertainty is unavoidable, in part because the scientific information used to develop estimates, such as the inventory of particulate matter emissions, will never be perfect or complete. According to the Academies, high uncertainty does not imply that action to promote or protect public health should be delayed, but rather that a comprehensive and rigorous assessment of uncertainty would improve policy decisions. Many of the recommendations of the National Academies to EPA are aimed at the agency developing a more robust uncertainty analysis by providing comprehensive, quantitative information about the uncertainties underlying the estimates of health benefits. A quantitative uncertainty analysis can, among other things, identify which sources of uncertainties have the greatest effect on the benefit estimates and assess the degree to which the uncertainties affect the estimates by specifying a range of estimates and the likelihood of attaining them. While EPA has taken steps toward a more robust uncertainty analysis, the agency has not assessed uncertainty in the quantitative manner

recommended by the National Academies. GAO believes it is important for EPA to continue to strengthen its uncertainty analysis. In evaluating estimates of health benefits, decision makers and the public can better assess the likelihood of achieving

such benefits if they are provided with qualitative and quantitative information about the underlying uncertainties.

Question 3. Mr. Stephenson, in your testimony (quote): "We note that continued commitment and dedication of resources will be needed if EPA is to fully implement the improvements recommended by the National Academies." However, EPA's 2006 budget proposes to reduce funding for the State and Local Air Quality Management Program which is used by states and localities to conduct monitoring among other things. How would this budget cut impact EPA's ability to address more of these recommendations?

Response. The proposed budget cuts could adversely impact EPA's ability to address more of the Academies recommendations, such as those concerning particulate matter emissions data. Emissions estimates are a key element in the analysis of potential health benefits, and EPA relies largely on state and local entities emissions monitoring data to develop estimates of emissions on a national scale. Thus, any reductions in emissions monitoring could affect EPA's ability to develop meaningful nationwide emissions estimates, increasing, for example, the uncertainty of such estimates. Moreover, as noted in our report, EPA has initiated long-term research and development to understand the relative toxicity of particulate matter components and to better evaluate the uncertainty of estimates of emissions. A reduction in monitoring data would likely limit EPA's progress to address these areas of research.

STATEMENT OF GEORGE THURSTON, ASSOCIATE PROFESSOR, NEW YORK UNIVERSITY, SCHOOL OF MEDICINE, DEPARTMENT OF ENVIRONMENTAL MEDICINE

I am George D. Thurston, a tenured Associate Professor of environmental Medicine at the New York University (NYU) School of medicine. In addition, I served as the Deputy Director of the NYU-EPA Particulate Matter (PM) Health Research Center for the past 4 years. My scientific research involves the investigation of the human health effects of air pollution. In this testimony, I will primarily address three factors that need to be considered in the EPA's proposed revisions to the particulate matter air quality standards. First, I will address the fact that we are now far more certain of the adverse impacts and biological mechanisms of PM health effects: most of the uncertainties raised at the time of the initial setting of the $PM_{2.5}$ standard are far better understood. Second, I will document that reducing ambient PM levels can and do result in significant reductions in the mortality risk associated with this pollutant. Finally, I will show that the adverse health impacts of PM air pollution extend below the current $PM_{2.5}$ standard, and that there is, therefore, a public health imperative to reduce the fine particle ($PM_{2.5}$) annual standard below 15 wg/m^3, consistent with the advice of the U.S. EPA's Clean Air Science Advisory Committee (CASAC).

Despite progress over the last few decades, Americans are still suffering from the adverse health effects of air pollution. The adverse health consequences of breathing air pollution are severe and well documented in the published medical and scientific literature. Over the past few decades, medical researchers examining air pollution and public health, including myself, have shown that air pollution is associated with a host of serious adverse human health effects, including: asthma attacks, heart attacks, hospital admissions, adverse birth outcomes, and premature death.

PM is one of the air pollutants most carefully studied in the last decade. Small particles can bypass the defensive mechanisms of the lung, and become lodged deep in the lung where they can cause a variety of health problems. Indeed, the latest evidence indicates that exposures cannot only cause respiratory damage, but also cardiac effects, including heart attacks. Moreover, long-term exposure to fine particles increases the risk of death, and has been estimated to take years from the life expectancy of people living in the most polluted cities, relative to those living in cleaner cities (Brunekreef, 1997).

The State of the science on particulate matter and health was thoroughly reviewed in the recently released U.S. EPA Criteria Document for Particulate Matter (U.S. EPA, 2004). Since the $PM_{2.5}$ standard was last set in 1997, more than 100 new published studies, taken together, collectively confirm the relationship between $PM_{2.5}$ pollution and severe adverse human health effects. In the process, this new research has eliminated many of the doubts that were raised in the past regarding the causality and size of the PM-health effects relationships, and has now provided plausible biological mechanisms for the serious impacts associated with PM exposure. As outlined in Figure 1, the PM research funded since the setting of the last $PM_{2.5}$ standard has collectively shown the existence of numerous biological pathways capable of causing damage in the human heart, lung, nervous system, and cir-

culatory system consistent with the health impacts found by the PM epidemiology studies upon which the $PM_{2.5}$ standard was set.

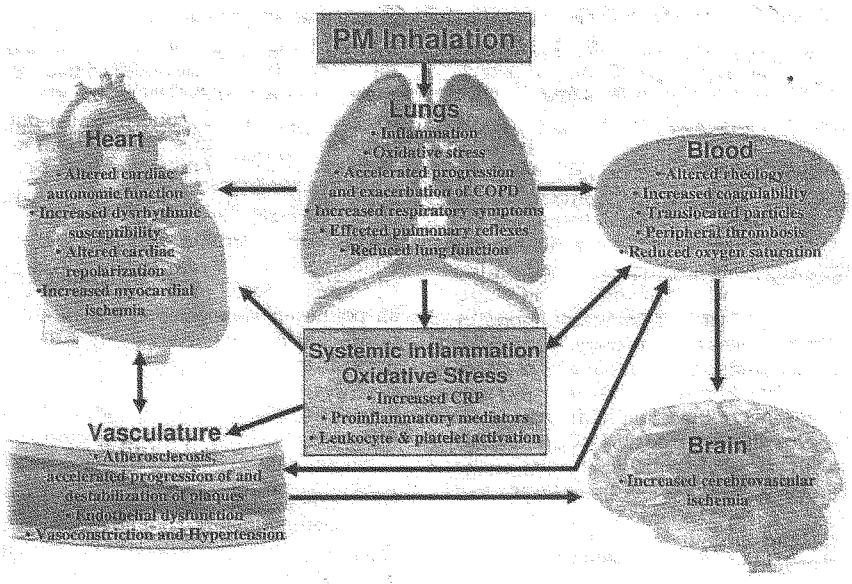

Figure 1. New Research Has Found Many New Pathways for PM Health Effects (Source: Pope & Dockery, 2006)

At the time of the last PM standard revisions, the largest landmark studies on particulate matter and death were the Harvard Six Cities Study (Dockery et al, 1993) and the American Cancer Society Study (Pope et al, 1995). The American Cancer Society study examined half a million people in over 150 metropolitan areas throughout the United States, finding a 17 percent greater risk of mortality between the city with the least PM and the city wit the highest levels of this particulate pollution. The results of these studies were challenged by industry, resulting in an independent reanalysis by the Health Effects Institute (HEI)—an organization funded by both industry and Government. The results of the HEI re-analyses have now confirmed the associations found by the original investigators, increasing our confidence in the results of these two already highly regarded studies of PM mortality.

Since the setting of the original $PM_{2.5}$ standard, more recent follow-up analyses of the Harvard and ACS studies have now considered longer records of time, and have confirmed and extended the conclusions from these two major studies. An extended analysis of the Harvard Six Cities Study (to include follow-up through 1990) has now shown that reductions in long-term ambient PM pollution results in concomitant reductions in the health risks associated with PM. As shown in Figure 2, large reductions in PM at four of the Harvard cities have resulted in likewise large reductions in the relative risk (RR) of mortality in those cities: Steubenville, OH(S), Harriman, TN(H), St. Louis, MO(L), and Watertown, MA(W). Other published studies have similarly found indications that reductions in ambient PM are associated with reduced mortality risk (e.g., Clancy et al., 2002). Thus, although we still carry very large health risks in the United States from our present levels of PM air pollution, amounting to tens of thousands of premature deaths per year, and although we still have a long way to go to have what can be called "clean air", recent research shows that the lowering of PM levels in the air is an effective way to improve public health.

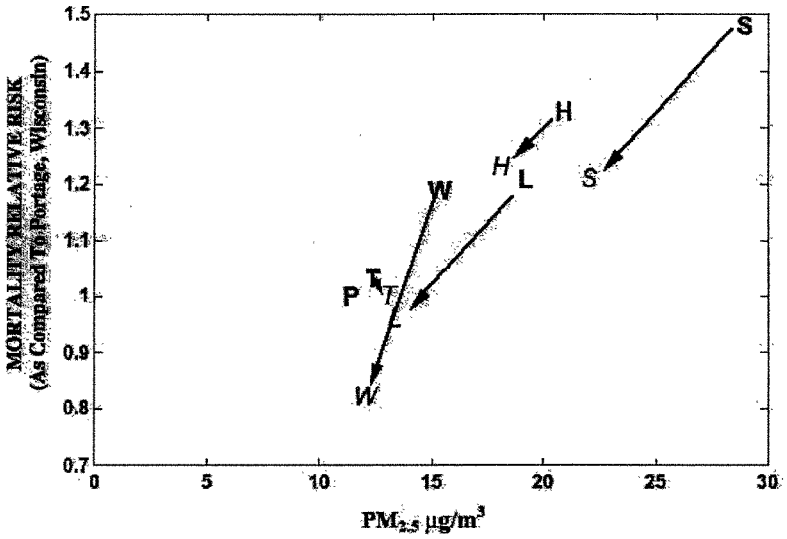

Figure 2. Reducing long-term PM exposure reduces mortality risk (Derived from Laden et al., 2006)

In addition, a recent National Institute of environmental Health Sciences (NIEHS)-funded extension of the ACS study, of which I was Principal Investigator, strengthens the original conclusions of the ACM study and, importantly, now links increased risk of lung cancer to long-term exposure to particulate matter (Pope et al, 2002). As seen in Figure 3, this recent JAMA study also clearly indicates that the risks from $PM_{2.5}$ exposure extend below 15 ug/m^3, supporting a reduction in the annual $PM_{2.5}$ standard at this time, consistent with the advice of the EPA's Clean Air Scientific Advisory Committee (Henderson, 2006).

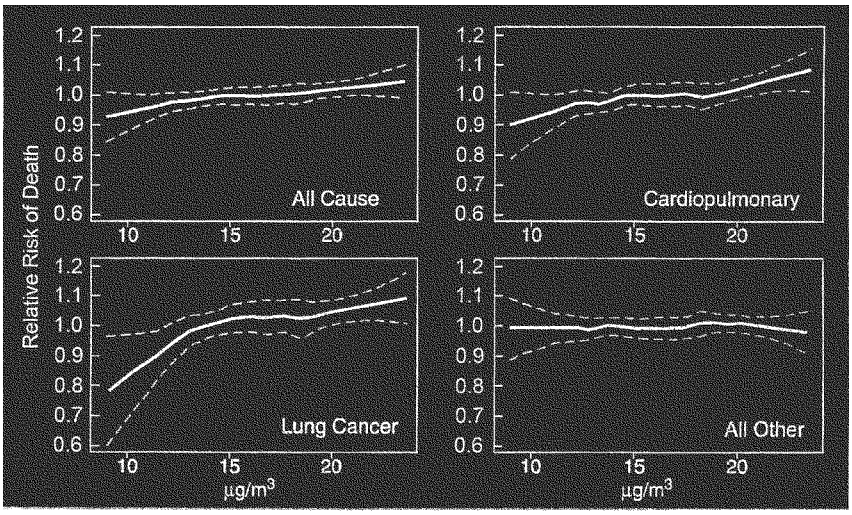

Figure 3. The extended ACS JAMA Study indicates that mortality effects extend well below the present fine particle standard of 15 *u*g/m^3 $PM_{2.5}$. (Pope et al, 2002).

In conclusion, since it was the level of uncertainty about PM biological mechanisms and effects at lower concentrations than 15 wg/m^3 that limited the standard to that level in 1997 (and not some specific acceptable level of health risk from PM),

and since new sound scientific studies have greatly reduced or resolved those uncertainties, then concern about the health of the public clearly indicates that the long-term PMPM$_{2.5}$ standard should now be reduced below 15 ug/m^3, consistent with the advice of CASAC.

Thank you for the opportunity to testify on this important issue.

RESPONSE BY GEORGE THURSTON TO AN ADDITIONAL QUESTION FROM SENATOR VOINOVICH

Question. Dr. Thurston, at the end of your testimony, you state the annual standard was limited to 15 not because of (quote): "some specific acceptable level of health risk from PM."

Am I correct that you are saying that you agree with Dr. Smith that the risk level identified in 1997 has gone down? I know that you do not think this should be the basis of setting the standard, but I am just looking for a yes or no answer on the level of risk.

Response. No, I do not agree with Dr. Smith. As I noted in my verbal testimony, the consensus "best-estimates" for the mortality effects of long-term exposure to PM have actually increased in recent years, relative to what the U.S. EPA has been assuming in their risk analyses. Indeed, the Pope et al. (2002) overall mortality effect estimate, as derived from the American Cancer Society Cancer Prevention II study cohort, is now thought to be an underestimate of the actual risk for the general U.S. population. In fact, preliminary results from the EPA's recent PM Expert Elicitation evaluation of PM risk indicate that the central effect estimate, as derived from a distillation of all available studies by experts, yield a $PM_{2.5}$ mortality effect (some 1 percent per ug/m^3 $PM_{2.5}$) that is roughly two thirds higher than given by the Pope et al. (2002) study. This further indicates that

lowering the long-term $PM_{2.5}$ standard below 15 ug/m^3 would yield significant public health benefits when implemented across the United States.

INTERNATIONAL

Technical Comments on the Proposed Rule for National Ambient Air Quality Standards for Particulate Matter

Prepared on behalf of the Utility Air Regulatory Group

Anne E. Smith, Ph.D.
CRA International
April 17, 2006 (with corrections on April 27, 2006)

I. Introduction and Summary of Key Points

EPA published its Proposed Rule ("PR") for the National Ambient Air Quality Standards for Particulate Matter on January 17, 2006.[1] This document contains my comments on the technical basis in the PR for the levels that EPA proposes for the $PM_{2.5}$ daily standard (*i.e.*, 35 $\mu g/m^3$ 98^{th} percentile 24-hour average $PM_{2.5}$) and for the $PM_{2.5}$ annual standard (*i.e.*, 15 $\mu g/m^3$ annual average $PM_{2.5}$).

EPA uses both a risk-based approach and an evidence-based approach to determine whether to tighten the $PM_{2.5}$ standards. It uses an evidence-based approach to support the specific proposed levels for the $PM_{2.5}$ standards. When one considers a more complete summary of the available epidemiological evidence than EPA offers in the PR, neither the risk analysis nor the evidence-based approach provides support for tightening either $PM_{2.5}$ standard. Furthermore, even if one were to accept the view that the daily standard must be tightened below current levels, a more complete summary of the available evidence than provided in the PR reveals that its evidence-based approach provides no technically-based guidance for where to set a standard.

The reasons that the quantitative risk analysis does not support a tightening of the $PM_{2.5}$ standards are: (1) the risk estimates are generally lower than they were in 1997, when the standards were first set; and (2) the overall robustness of statistical significance associated with the quantitative estimates has fallen dramatically since 1997, driven mainly by greater evidence and acknowledgment of the importance of modeling uncertainties. This eroding confidence directly reflects an eroding basis in the epidemiological evidence itself; therefore, one cannot argue that overall evidence supports a tightened standard even if the quantitative risk estimates do not. Additionally, the quantitative risk analysis that is provided is biased upwards because it does not incorporate the larger body of quantitative findings regarding model uncertainty. The majority of the evidence that EPA's risk analysis ignores would produce lower risk estimates, and imply greater likelihood that there is no $PM_{2.5}$ effect at all. I substantiate these statements in Part II of this document.

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[1] Federal Register, Vol. 71 (10), pp. 2620-2708.

EPA uses an evidence-based approach to attempt to identify a justifiable level below 65 $\mu g/m^3$ for setting a daily standard. EPA's application of this approach, however, is incomplete in that it relies on only a subset of the actual available literature, and it does not provide evaluation of the quality of evidence in each study that it does cite. When these gaps are filled, and the results are presented in a more structured manner, it becomes clear that EPA's line of reasoning for setting the standard at 35 $\mu g/m^3$ is not supportable. Part III of this document provides the detailed analysis demonstrating these points.

Finally, the PR argues that the recent literature has found that estimated associations between $PM_{2.5}$ and health endpoints are generally robust to inclusion of gaseous pollutants in the epidemiological model. This statement is a linchpin in the PR's case that a causal role for $PM_{2.5}$ has been more strongly established since 1997. Unfortunately, it is also an incorrect conclusion based on only a subset of the full body of $PM_{2.5}$ epidemiological literature. In Part IV, I review and summarize the evidence on the impact of considering gaseous pollutants simultaneously with $PM_{2.5}$ in epidemiological modeling. My review reveals that statistical significance of $PM_{2.5}$-health associations is *not* robust when one or more gaseous pollutants are included. My review also finds that this sensitivity does not appear to be caused by problems of multicollinearity. Thus, EPA should eliminate this conclusion, and revise its analysis concerning the public health risk from $PM_{2.5}$ without reference to such a conclusion.

II. Quantitative Risk Analysis Does Not Support Tightening the Standards

EPA has decided that its quantitative risk analysis cannot be used to decide where to set a standard.[2] This is a reasonable conclusion because, in the absence of any specific knowledge of where an effects threshold may lie, the risk analysis will always produce linearly declining amounts of risk for each incremental reduction in the level of the standard until background concentrations are attained. However, the risk analysis also fails to provide a case for tightening the current standards, both in its explicit results, and also because EPA's risk analysis incompletely represents all of the relevant quantitative evidence. The PR does not fully recognize these two points, and thus makes a technically unsound case that the risk analysis does provide support for tightening the daily standard. Although EPA has not proposed to tighten the annual standard, it is also the case that current evidence would not support such a reduction.

Quantitative Risk Estimates Are Lower Now Than When the Current Standards Were Set, and Confidence in the Risk Associations Is Also Reduced

A key insight from EPA's risk analysis is that current quantitative estimates of risk levels that remain at exact attainment of the current $PM_{2.5}$ standards are lower now than they were when those standards were set.[3] This result occurs even under EPA's "base case" assumptions, which overstate the current evidence on risk levels. Risk estimates would be lower still under most of the alternative set of assumptions that EPA does not use in its "base case" calculations. If risk levels are estimated to be lower than they were at the time that standards were originally set then, *ceteris paribus*, one cannot make a quantitative case for tightening the $PM_{2.5}$ daily standard.[4] This outcome of the risk analysis is mentioned briefly in the PR[5] but the extent of the change in point estimates of risk since 1997 is understated:

> "With respect to short-term exposure mortality and morbidity ... [c]omparing the risk estimates for the only two specific locations that were included in both the prior and current assessments, the magnitude of the estimates associated with just meeting the current annual standard, in terms of percentage of total incidence, is similar in one of the locations (Philadelphia) and the current estimate is lower in the other location (Los Angeles). ... With respect to long-term exposure mortality risk estimates, the estimates in terms of percentage of total incidence are very similar for the two specific locations included in both the prior and current assessments."[6]

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[2] PR, p. 2648.

[3] These risk estimates are due to lower epidemiological estimates of the relative risk of $PM_{2.5}$, and are *not* lower due to reduced air pollution since 1997. (The air quality used to make these risk estimates is effectively the same now as in 1997 for the simulation of exact attainment of the current standards on which they are based.)

[4] As EPA notes, even if risk estimates are lower, a case might still be made to tighten the standards if the confidence in the risk estimates is increased. As I will explain next, the technical evidence now available does not engender greater this greater confidence.

[5] It was not mentioned at all in the Staff Paper (EPA (2005)), or in the risk analysis (Post *et al.* (2005)).

[6] PR, p. 2640.

EPA did not report these comparative results of the risk analysis in any of the documents leading up to the PR,[7] and even the PR does not clearly explain the policy-relevant facts. The following sections provide the facts behind the above quote, and reveal that the degree of reduction in quantitative risk estimates is more pronounced than a mere comparison of EPA's base case risk estimates would imply. It is also possible to infer that reduced risks have occurred for all but one of the new cities that have been included in the current risk analysis.

Short-term Mortality Risk. In 1997, EPA performed a risk analysis for two cities, Los Angeles and Philadelphia. EPA based its estimates of short-term mortality risk for both cities on a statistically significant relative risk estimate from Schwartz, Dockery and Neas (1996), while I will refer to hereafter as "SDN". Neither of the two cities had been represented in SDN, but this was the only study of short-term mortality risk associated with $PM_{2.5}$ available at the time. Since then, part of the newly available body of evidence includes short-term mortality studies specific to these two cities: Moolgavkar (2003) for Los Angeles and Lipfert *et al.* (2000a) for Philadelphia. EPA has relied on these two new studies for its current risk analysis for these two cities.

Figure 1 (for Los Angeles) and Figure 2 (for Philadelphia) compare the 1997 short-term mortality risk estimate (and associated 95% statistical confidence ranges) to risk estimates supported by the two new studies. These are risk estimates for ambient $PM_{2.5}$ levels that are just attaining the current standards. In both figures, the leftmost vertical bar (shown in red) is the short-term percent mortality incidence estimated in 1997, based on SDN (as noted on the graph under that bar). One can see that the percent incidence attributed to $PM_{2.5}$ in 1997 was about 1.7% for Los Angeles and 1.5% for Philadelphia.

All of the blue bars (*i.e.*, all of the bars except for the leftmost one) are based on relative risk estimates from the newly available study for each city. There are multiple blue bars because these new studies reported results of $PM_{2.5}$ relative risk estimates using many different model formulations. The variations in models included use of GAM versus GLM methods of estimation, varying degrees of smoothing for time and weather, and use of single-pollutant ("1-P") versus two-pollutant ("2-P") formulations that included one of the gaseous co-pollutants. Of all the alternative estimates in each study, EPA's base case risk estimates use only a single one, which is reflected in the leftmost blue bar, positioned to the left of a grey dashed divider line, next to the red bar reflecting the 1997 estimate. The two bars to the left of the dashed divider line thus provide a comparison of EPA's base case risk estimates from the 1997 and current risk analyses. They imply a large decrease in Los Angeles (from 1.7% to 0.5%) and a modest increase in Philadelphia (from 1.5% to 2.2%).

[7] It was, however, documented in my comments to EPA on the August 2003 draft Risk Analysis, and on the January 2005 second drafts of the Staff Paper Risk Analysis. (Smith (2003, 2005)). The exact levels of risk reported here have changed slightly since these drafts, and correspond to those in the final risk analysis and Staff Papers of June 2005.

Figure 1. Comparison of Short-term Mortality Risk Estimates in 1997 and Now When Ambient $PM_{2.5}$ is Just Attaining the Current $PM_{2.5}$ Standards – Los Angeles

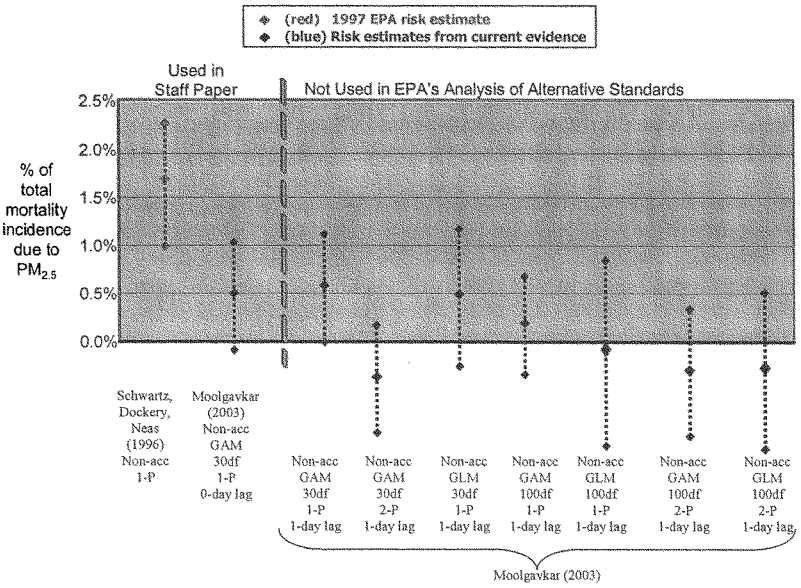
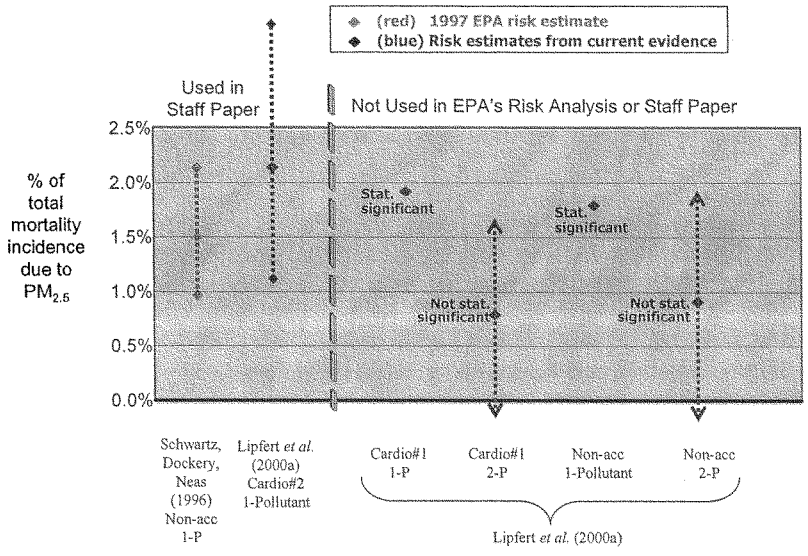


Figure 2. Comparison of Short-term Mortality Risk Estimates in 1997 and Now When Ambient $PM_{2.5}$ is Just Attaining the Current $PM_{2.5}$ Standards – Philadelphia



The additional lines to the right of the dashed divider line reflect the full range of new information that has been excluded from EPA's current base case risk analysis results. As can be seen, most of the alternative available risk estimates are much lower than the single one that EPA chose to use for its base case. Further, all of the alternative estimates using 2-P models are not only much lower in risk, but statistically insignificant. (In each of these cases, the gaseous second pollutant in the model *was* statistically significant.) In both papers, the authors concluded that the 2-P results indicated that the culprit pollutant appeared to be a gaseous pollutant rather than $PM_{2.5}$, and that use of any of their 1-P model results would *overstate* the case for a $PM_{2.5}$-mortality association.

Thus, a full comparison of the newly available risk information to that which was available in 1997 indicates a substantial reduction in estimated risk levels. Further, this review reveals that EPA's current base case risk estimates substantially overstate the short-term $PM_{2.5}$ mortality risks that the newly studies imply, because they rely solely on 1-P model results that the authors themselves discredit.

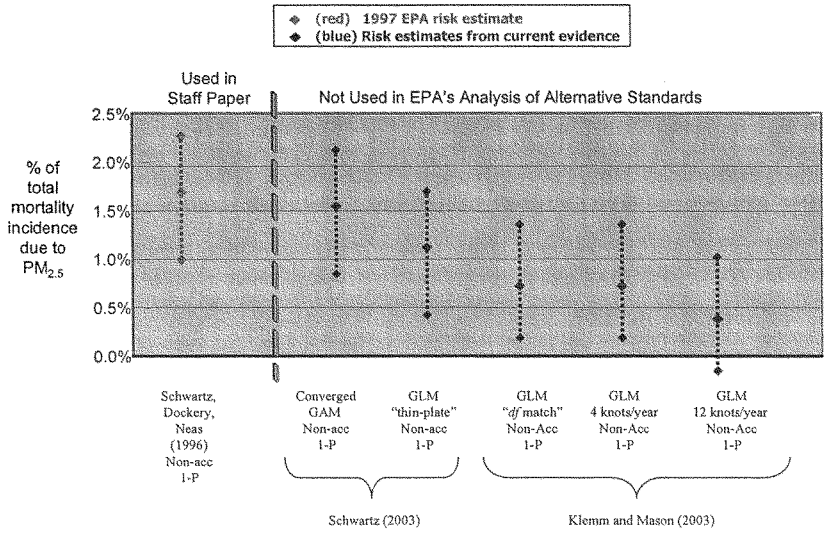
Los Angeles and Philadelphia are given special focus in the comparison of risk analysis findings because those were the only two cities for which risk estimates were developed in the 1997 analysis. However, it is quite feasible to determine what the percent mortality incidence would have been for other cities in the current risk analysis if they *had* been included in a 1997 risk analysis. This is because risk estimates in all of those cities would have also been based on SDN, just as they were for Los Angeles and Philadelphia.

Even if current risk estimates for the other cities were still to be based on the database used by SDN, they would be lower now than in 1997 as a result of reanalyses performed when correcting the GAM statistical error in SDN. The newly available studies replacing SDN are Schwartz (2003a) and Klemm and Mason (2003). Both new studies found that the relative risks in the original SDN paper fell as linear methods of estimation (GLM) were used to replace the GAM method, and as temporal controls were enhanced. Indeed, Klemm and Mason reported that the relative risk estimates became statistically insignificant in their most highly controlled formulations. Figure 3 shows how much risk estimates and confidence intervals for this dataset have declined as a result of the newly available results for the database used in SDN.[8]

St. Louis and Boston are two cities in the current risk analysis for which Schwartz (2003a) and Klemm and Mason (2003) are used, because these cities are among the "Six Cities" that are analyzed in these studies. Their individual city-specific estimates were affected in a similar manner to that depicted in Figure 3, although St. Louis was affected more, with all of the GLM findings in Klemm and Mason (2003) producing statistically insignificant associations.

[8] The figure shows results for Los Angeles (again, showing percent mortality incidence when just attaining the current standards), using the combined cities estimates. Although absolute levels of incidence will vary slightly from city to city, the *relative* change in estimated risk levels would be the same in any city to which these study results might be applied.

Figure 3. Comparison of Risk Estimates Based on SDN in 1997 to Those Based on Reanalyses of SDN Available Since 1997.
(The figure reflects the estimated mortality incidence just attaining the $PM_{2.5}$ standards in Los Angeles, but the relative pattern in the risk estimates would be the same for any city for which risk estimates might be based on these studies. The estimates are based on the combined cities results, but the patterns are very similar for each of the six individual cities in the studies as well.)



The remaining cities for which short-term mortality risks are provided in the current risk analysis are Phoenix, Detroit, Pittsburgh, and San Jose. There is a newly available city-specific study for each of these four cities.[9] The single base case relative risk estimate that EPA uses for three of these cities in its current risk analysis is lower than the relative risk estimate it would have used in 1997 (*i.e.*, the SDN combined cities result). The sole exception is San Jose, based on a risk estimate from Fairley (2003). Importantly, even the base case risk estimate for two of those three cities is now statistically insignificant, whereas the estimate that would have been produced in 1997 based on SDN would have been statistically significant.

Table 1 summarizes the overall state of newly available evidence used in the current risk analysis, as compared to the statistically significant estimate that was available for the 1997 risk analysis. For short-term $PM_{2.5}$ mortality risk, EPA's base case point estimates of risk are lower in six of the eight cities in the current risk analysis, and many of those base case estimates are themselves statistically insignificant. For the remaining two cities

[9] These are Mar *et al.* (2003), Ito (2003), Chock *et al.* (2000) and Fairley (2003), respectively.

(Philadelphia and San Jose), other model results in the source studies would, however, produce lower risk estimates, if used. Furthermore, *none* of the newly available studies for these eight cities finds a $PM_{2.5}$-mortality association that is statistically significant to all of the formulations that are reported in those studies. This is reflected in the last column of Table 1, that none of the cities' risk estimates remain statistically significant under all the model outcomes that are reported for each city.

Table 1. Summary of Declining $PM_{2.5}$ Risk Estimates and Reduced Confidence in Statistical Significance of Current Risk Estimates Compared to 1997.

		Did EPA's risk estimate rise or fall since 1997?(*)	Is EPA's risk estimate statistically significant?	Is significance of estimate robust to alternative model choices?
Short-term risk	Philadelphia	Up	Significant	Not robust
	Los Angeles	Down	Insignificant	Not robust
	Phoenix	Down	Significant	Not robust
	St. Louis	Down	Significant	Not robust
	Boston	Down	Significant	Not robust
	Detroit	Down	Insignificant	Not robust
	Pittsburgh	Down	Insignificant	Not robust
	San Jose	Up	Significant	Not robust
Long-term risk	All Cities	Down	Significant	Not robust

(*) For the short-term risks, the 1997 estimate is assumed to be the SDN "combined" for all cities except for Boston and St. Louis, for which it is the SDN city-specific estimate. Current estimate is the risk coefficient used in *Staff Paper* to estimate short-term mortality risk reduction under alternative standards (*e.g.*, Figure 5-2).

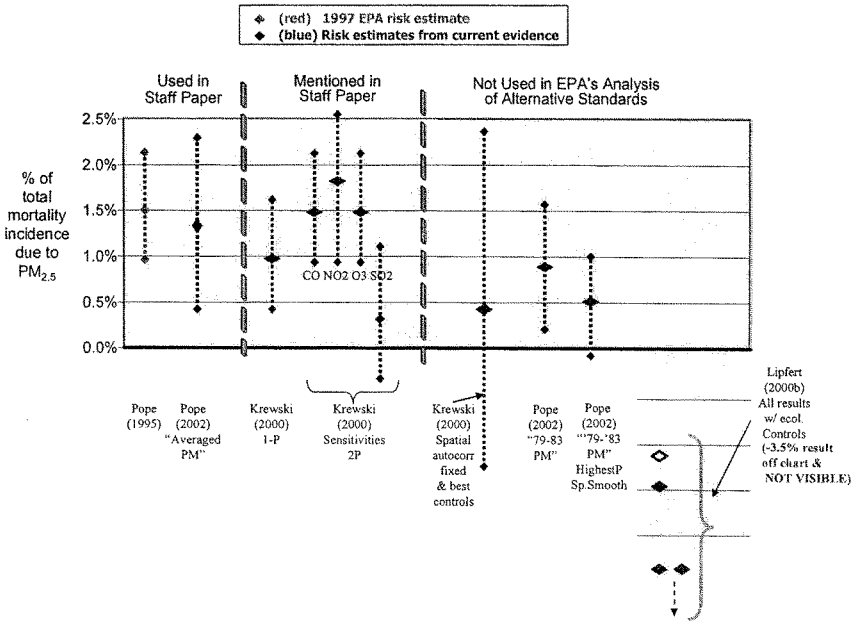
Thus, the evidence reflects a very strong case that short-term mortality risks estimates are lower now than they were in 1997. EPA's statement on this point, quoted above, substantially understates this trend in risk estimates since the current standard were set. The quantitative risk analysis for short-term mortality from $PM_{2.5}$ does not support a tightening of the $PM_{2.5}$ standards. Instead, the quantitative risk analysis reveals a consistent pattern of decreased risk even when using the "highest" and "most significant" estimates from each of the newly available epidemiological studies. Additionally, these same new studies provide strong evidence that the $PM_{2.5}$-mortality associations are not robustly statistical significant (*i.e.*, statistical significance is eliminated in the face of a variety of reasonable alternative statistical modeling methods and formulations.)

Long-term Mortality Risk. Lower risk levels and eroding confidence in the underlying statistical evidence also apply to long-term mortality risk estimates, as Table 1 also shows. EPA's statement, quoted above, that the long-term risk estimates are "very similar" to those in 1997 is not consistent with the facts. EPA's base case long-term risk estimates rely only on evidence in a dataset that was available in 1997, the American Cancer Society ("ACS") cohort. Although parts of the PR recognize that reanalyses of the ACS dataset have revealed important uncertainties in its statistical associations for $PM_{2.5}$, EPA's base case risk analysis ignores the major impact these sensitivities have on the level of risk and associated confidence levels. Additionally, even the sensitivity analyses in the risk analysis completely ignore one newly available study, Lipfert *et al.* (2000b), that reports $PM_{2.5}$ health associations in a cohort that had not even been studied as of 1997. EPA chooses to give this study no weight in its risk analysis, yet it provides some important new evidence that should at least be considered in an evaluation of trends in size of effect and confidence levels that can be assigned to the long-term mortality associations.

Figure 4 provides a graphical summary of the past and newly available evidence on quantitative long-term mortality risks. The percent risk incidence data in this figure also pertain to $PM_{2.5}$ levels at exact attainment of the current standards, as for the short-term mortality figures above. Risk estimates in Figure 4 are based on Los Angeles, but the patterns would be identical for any city in the U.S. because long-term mortality risk estimates are based on a single relative risk estimate applicable to all cities.[10] As with the previous figures for short-term mortality risk, the leftmost vertical bar (in red) reflects the level of risk that was estimated for just-attaining the current $PM_{2.5}$ standards in the 1997 risk analysis: about 1.5% of total mortality incidence. As noted below that bar, the 1997 estimate was based on the relative risk in Pope *et al.* (1995), which studied the ACS cohort. The first blue bar, coupled in the left segment of the figure with the 1997 estimate, reflects the current risk analysis's base case estimate of mortality risk: about 1.3%. As noted on the figure, this base case risk estimate comes from Pope *et al.* (2002), which also used the ACS cohort, but with data extended since the 1995 study. (It is, specifically, the relative risk based on "averaged PM" concentrations from that paper.) These two risk analysis estimates reveal a slight decline in long-term risk estimates since 1997. However, the remainder of the estimates presented in Figure 4 show the broader body of evidence. The broader body of evidence supports a conclusion that long-term risk estimates are substantially lower today than in 1997. It also shows that much less confidence can be assigned to long-term $PM_{2.5}$-mortality associations now than in 1997. The basis for these conclusions is discussed in detail below.

[10] This is because long-term risk studies are performed in a cross-sectional manner, by comparing mortality risks to pollution levels across cities. The resulting estimate is a single relative risk estimate *across* the cities in the study, rather than a different relative risk estimate for each city, as one obtains in the time-series type of study that produces a short-term mortality risk estimate.

Figure 4. Summary of Long-Term Risk Estimates Used in 1997 and Current Risk Analyses, and Comparison with Alternative Newly Available Estimates



Current long-term risk incidences are based on an LML value adjusted for comparability to 1997 estimates; All bars labeled "Krewski" use ACS-based risk estimates in Krewski *et al.* (2000); All examples are "non-accidental" mortality regressions. These results are based on Los Angeles, but the pattern is the same for Philadelphia, and all other cities.

The risk estimates shown in the middle segment of Figure 4 (between the two grey dashed divider lines) show the implications of including controls for of gaseous co-pollutants when analyzing the $PM_{2.5}$-mortality associations in the ACS cohort data (*i.e.*, "2-P" model results). This 2-P sensitivity analysis was performed in the Krewski *et al.* (2000) reanalyses of Pope *et al.* (1995). The first bar in this segment is the 1-P result in the reanalysis, and the next set of 4 bars reflects the comparable $PM_{2.5}$ risk estimate from 2-P formulations, which included CO, NO_2, O_3, or SO_2, respectively. These sensitivity analyses, which are mentioned in the PR, reveal that the $PM_{2.5}$ association is not robust when SO_2 is included in the regression.[11] The size of the $PM_{2.5}$ risk estimate falls

[11] The PR notes at p. 2631 that inclusion of SO_2 in the analysis "decreased the size of the effect estimates for PM2.5 to one-sixth of its original value and for sulfates to less than one-third of its original value." The PR notes this sensitivity again at p. 2634 and p. 2652.

dramatically relative to any of the 1-P risk estimates, and it becomes statistically insignificant.

Remarkably, despite this important finding in the reanalyses of 2000, Pope *et al.* (2002) still did not report any 2-P results using SO_2. This is especially remarkable because that paper *does* report that SO_2 has a statistically significant association with long-term mortality in its own 1-P regression, yet the authors simply do not present any results where $PM_{2.5}$ and SO_2 had been considered simultaneously. Even EPA notes this as an unusual omission, "[b]ecause the correlation coefficient between $PM_{2.5}$ and SO_2 was 0.50 in the ACS data, in this view it is plausible to believe that the independent effects of the two pollutants could be disentangled with additional study."[12] Thus, the evidence of this non-robustness is observable only in the older study of Krewski *et al* (2000), which EPA chooses to ignore in its base case risk analysis in favor of the more recent, but less comprehensive results in Pope *et al.* (2002).

Despite this dramatic sensitivity to SO_2, EPA's base case risk estimate relies on the 1-P formulation from Pope *et al.* (2002) and, elsewhere in the PR, EPA contends that risk estimates are "generally robust" to inclusion of gaseous pollutants.[13] The result is that both the risk analysis and the PR overstate the evidence in favor of a long-term mortality association for $PM_{2.5}$.

The rightmost segment of Figure 4 provides several additional risk estimates that are never mentioned in EPA's quantitative risk analysis even as sensitivity analyses. Aspects of these other results are only briefly noted in the PR's discussion of the overall evidence. When included in this figure, enabling a direct quantitative comparison to the assumptions used in the quantitative risk analysis, their implications for an eroding level of confidence (and yet lower risk estimates) become far clearer than one can ascertain from the text of the PR.

The first vertical bar in the rightmost segment of Figure 4 reflects the $PM_{2.5}$ risk estimates in the Krewski *et al.* (2000) reanalyses after removing undesirable spatial autocorrelation in the base case estimates from the ACS cohort, and simultaneously controlling for SO_2 and other important explanatory variables that were not included in relative risk estimates used in the risk analysis. The resulting reduction in risk level and statistical significance is so dramatic that it calls into question the causal interpretation of any of the other ACS-based relative risk estimates. Although Pope *et al.* (2002) did not provide such a 2-P formulation, it did report a 1-P example of the impacts of adding spatial controls. The effect, which can been seen by comparing the next two vertical bars to the right, was again to make the $PM_{2.5}$ relative risk estimate statistically insignificant. (The PR obscures this finding when it states that "Pope et al. (2002) reported that effect estimates were not highly sensitive to spatial smoothing approaches intended to address spatial autocorrelation."[14]) One can only surmise what the impact would have been on the Pope

[12] PR, p. 2652.
[13] PR, p. 2660.
[14] PR, p. 2652.

et al. (2002) $PM_{2.5}$ risk estimate if both spatial controls and SO_2 had been simultaneously included in that paper.

Finally, the far right portion of Figure 4 presents the $PM_{2.5}$ risk results from Lipfert *et al.* (2000b), which relies on an entirely different cohort, the "Veterans Cohort." This study was formally excluded from any part of the quantitative risk analysis, and is largely downplayed in the PR. The PR says only the following about this new long-term study:

> "In addition, one new set of analyses was done using subsets of PM exposure and mortality time periods and data from a Veterans Administration (VA) cohort of hypertensive men. The investigators report inconsistent and largely nonsignificant associations between PM exposure (including, depending on availability, TSP, PM_{10}, $PM_{2.5}$, PM_{15} and $PM_{15\text{-}2.5}$) and mortality."[15]

One can see from Figure 4, however, that EPA's characterization of this study as having "inconsistent" findings is *not* that the results within this study are inconsistent, but that this study's findings *are completely inconsistent with those that EPA has chosen to rely on*. In fact, the Veterans Cohort dataset contains *consistently negative associations* between $PM_{2.5}$ and long-term mortality. Furthermore, the PR is incorrect in stating that those associations are "largely nonsignificant." All but one of the associations between mortality and $PM_{2.5}$ in this paper are *significant in the negative direction* (reflected by a solid point estimate in Figure 4, as the numerical confidence ranges are not provided in the paper). EPA feels it should rely primarily on cohorts that had already been studied in 1997, and which have been reanalyzed since 1997. Regardless of this, EPA should still acknowledge that evidence from study of the newly available cohort casts greater uncertainty on the long-term $PM_{2.5}$-mortality association.

Figure 5 illustrates a final aspect of the newly available evidence that further clouds confidence in relative risk findings reported for $PM_{2.5}$-mortality associations based on the ACS and Six-Cities cohorts. This is the fact that the association between $PM_{2.5}$ and mortality in those two cohorts is entirely attributable to the individuals within those cohorts that have a high school education or less. The PR does describe this finding, but it is not reflected in any way in the quantitative risk analysis. There is no statistical significance in the association for individuals within these two cohorts that have more than a high school level education. The point estimate of relative risk for these individuals is effectively unity. This pattern, first identified by Krewski *et al.* (2000) in their reanalysis report, appears in *both* of the cohorts that EPA has chosen to emphasize, and to rely on for its quantitative risk estimates. The effect has persisted into the extended dataset of Pope *et al.* (2002). There are a number of hypotheses that can be offered for what this means, but all of these hypotheses lead to conclusions that either the relative risk estimates being used in the risk analysis are biased, or that the association with $PM_{2.5}$ is actually due to some other confounder, and is not causal.

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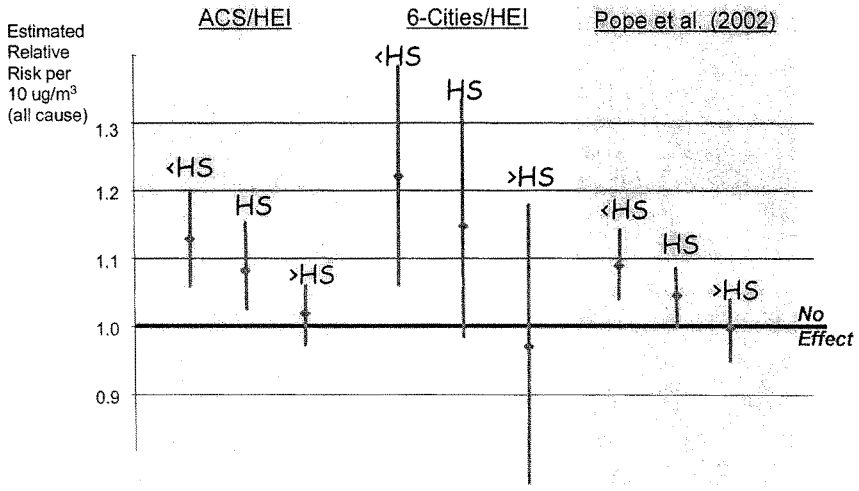
[15] PR, p. 2632.

Figure 5. Persistent Pattern of Long-Term Mortality Associations Being Applicable only to Individuals in Cohorts Who Have Lower Educational Levels



"ACS/HEI" refers to the reanalysis of the ACS cohort in Krewski *et al.* (2000). The relative risks shown were taken from Table ES-3 therein, and converted from units "per 24.5 $\mu g/m^{3n}$ to relative risk per 10 $\mu g/m^3$. The label "6-Cities/HEI" refers to the reanalysis of the Six-Cities cohort in Krewski *et al.* (2000). The relative risks were taken from Table ES-3 therein, and converted from units "per 18.6 $\mu g/m^{3n}$ to per 10 $\mu g/m^3$. The results from Pope *et al.* (2002) were taken from Figure 4A of that paper.

Summary. For both long-term and short-term mortality risks, the newly available evidence reveals decreased risk levels, and heightened uncertainty about the nature of the associations, compared to the body of evidence that was available in 1997. This is revealed even in EPA's quantitative risk analysis results, even though that analysis is biased upwards by selective use of model results that have the largest and most significant findings in each source study. This bias is enabled by EPA's decisions to rely only on 1-P results, its use of GAM-based analyses rather than GLM-based estimates, and its use of model formulations with the least amount of controls of those reported in each paper.

EPA has elected not to tighten the current annual standard in light of its qualitative acknowledgment of these types of uncertainties, but the PR provides a weaker case for this reasonable conclusion it actually could make. The quantitative risk analysis and related new epidemiological evidence similarly fail to provide a technical basis for tightening the daily $PM_{2.5}$ standard, yet the PR does not make this case even weakly. Instead, the PR obscures this case by not providing a balanced discussion of the sensitivities in the new evidence on short-term mortality associations with $PM_{2.5}$. The PR

also does not fully reveal the extent of the decline in EPA's quantitative risk estimates since 1997.

An Integrated Uncertainty Analysis in the Risk Analysis Would Reveal the Eroding Confidence Levels

EPA is clearly aware that there is enormous uncertainty in the newly available evidence. The PR solicits comments on its methodology for evaluating the uncertainty and significance of risks to public health, and specifically on "methods and approaches for conducting a more formalized uncertainty analysis."[16]

EPA's methodology has been to rely on single "base case" estimates in a quantitative risk analysis, combined with occasional references to sensitivities in these results. The figures provided above provide a quite different story, revealing that EPA's current methodology has resulted in overstatement of the risk levels and overstatement of confidence in $PM_{2.5}$-health associations. Lack of a clear comparison to earlier risk estimates also obscures what is a pronounced trend towards lower risk estimates and eroded confidence levels. The net effect is that EPA's case to support tightening the daily standard is not supportable with the full evidence. This tendency toward overstatement of risks in EPA's approaches to handling uncertainty can be averted merely by more complete and clear representation of the evidence, without any formal uncertainty analysis. As the section above has demonstrated, merely providing complete and quantitative information from the full body of epidemiological evidence can provide a far clearer synopsis of the evolution of confidence in the associations.

At the same time, a carefully conducted synthesis of the evidence into an integrated uncertainty analysis of the quantitative risk estimates could also help in standard-setting deliberations. I have provided detailed comments on such an approach and examples using the currently available evidence in previous written comments to EPA, Smith (2003, 2005) which I incorporate here by reference. Figure 6 provides an example of how different the information resulting from an integrated uncertainty analysis can be from the "base case" approach that is the hallmark of EPA's analysis. This figure is taken from my earlier written comments to EPA, and its derivation is documented there in this set of comments. I will only discuss its interpretation and implications.

The histograms in Figure 6 represent full probability distributions from an integrated uncertainty analysis that weights EPA base case models as well as others from the full body of relevant literature.[17] They can be interpreted as follows. The x-axis is the percent incidence of long-term mortality attributed to $PM_{2.5}$ in risk calculations (the same metric as the y-axis in Figure 4).[18] Each bar of the histogram reports the probability that

[16] PR, p. 2653.

[17] Although the details of which models are included is documented in Smith (2003), briefly, they include alternative models shown in Figure 4 as well as others, such as those from the Six-Cities long-term cohort.

[18] In this example, however, the percent incidence is that associated with as-is $PM_{2.5}$ rather than with $PM_{2.5}$ at exact attainment of the current $PM_{2.5}$ standards. Hence the incidence levels are somewhat higher than they were in Figure 4.

the true risk falls in the range of percent incidence that the bar sits over. The y-axis is the probability associated with each bar. The first bar is colored differently from others because it reflects the likelihood that there is no $PM_{2.5}$ risk at all. That is, its height reflects the probability that risk is exactly 0.[19] The yellow bars show the probability of various levels of positive $PM_{2.5}$ risk, expressed for ranges of risk levels. For example, the leftmost yellow bar shows the probability that the percent incidence lies in the range greater than 0% and less than or equal to 2%, and is positioned in the center of that range. The next bar reflects risks greater than 2% and less than or equal to 4%.

The blue horizontal lines in Figure 6 show the ranges of the 95% confidence intervals that EPA reports for its base case estimates of as-is risk for these two cities.[20] These are based on just the one model from Pope *et al.* (2002) that EPA has selected for its base case.[21] EPA's base case estimates show a significant effect in both cities at as-is $PM_{2.5}$ levels (*i.e.* no part of the EPA confidence intervals includes 0 percent incidence, where the red bar is located). These intervals reflect only the statistical variance associated with the underlying relative risk estimate selected from Pope *et al.* (2002), which was statistically significant.

As this section has demonstrated, many other relative risk estimates do exist that are *not* statistically significant, and the integrated uncertainty analysis incorporates their effect as well as the effect of the statistically significant ones such as EPA uses for its base case estimates. These other estimates account for the non-zero probability of "no effect" (the red bar of the integrated uncertainty analysis probability distribution.)

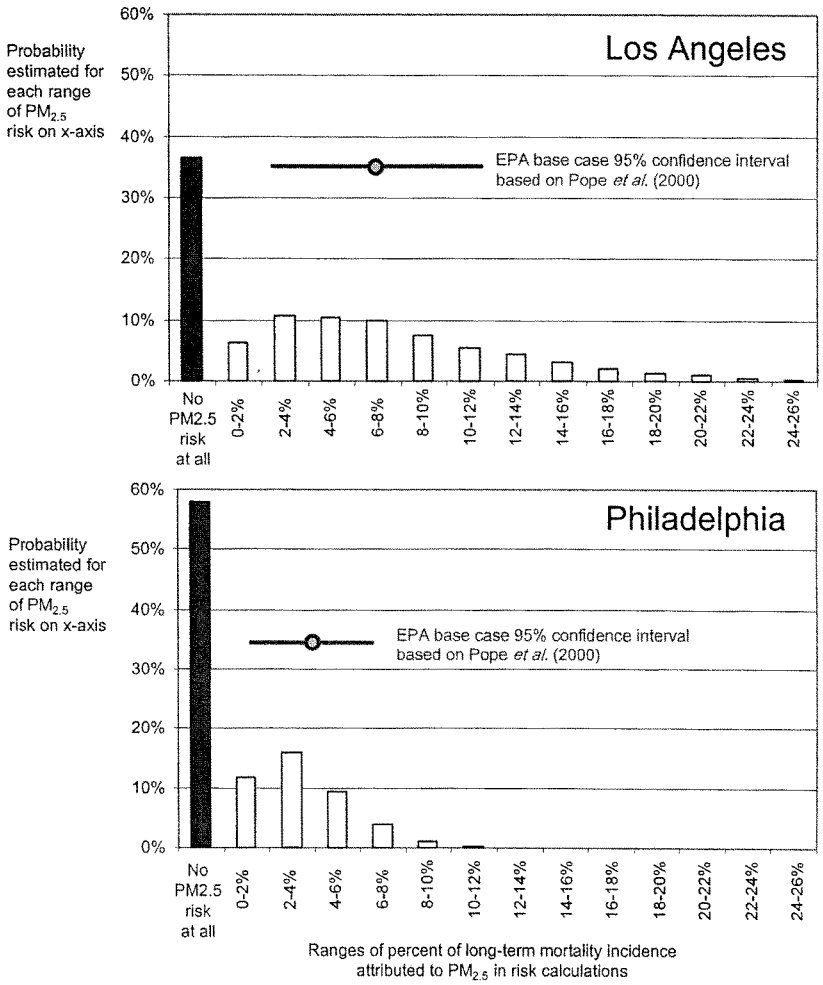
[19] Technically, this bar reflects a discrete pulse of probability associated with the single risk value of 0, whereas all other parts of the probability distribution are continuous, with zero probability for any single risk value.

[20] Technically, there is also a probability distribution over these ranges, which is a normal (bell-shaped) distribution centered over the point estimate that is shown as the circle on each bar. Only the ranges are shown here, for simplicity.

[21] EPA reports these two as-is risk ranges in Post *et al.* (2005), Exhibit 7.2, p. 85.

Figure 6. Examples of Probability Distributions on Percent Incidence of Mortality Due to Long-Term Exposure to As-Is $PM_{2.5}$ Resulting from an Integrated Uncertainty Analysis, Compared to "Confidence Intervals" Resulting from EPA's Approach of Relying on Base Case Estimates Only.

The blue horizontal lines reflect the EPA base case as-is risk estimate's 95% statistical confidence interval, based on Pope *et al.* (2002). The circle reflects EPA's base case point estimate of as-is mortality risk. The histogram provides the full probability distribution from an integrated uncertainty analysis that weights the EPA-selected model with others from the full body of relevant literature. The red bar reflects the likelihood of "no $PM_{2.5}$ risk" and yellow bars reflect relative likelihood of various levels of positive $PM_{2.5}$ risk. Documentation of analysis provided in Smith (2003).



One benefit of a more explicit, probabilistic approach is therefore that it provides a more complete and unbiased summary of the overall evidence than can be obtained with a deterministic approach that emphasizes results from a single selected base case model. The most dramatic effect of the integrated uncertainty analysis approach is how much probability is attributed to the outcome that there is no increased mortality risk at all from $PM_{2.5}$. It is over 35% for as-is conditions in Los Angeles, and over 55% for as-is conditions in Philadelphia.[22] In contrast, the EPA method of reliance on base case model results suggests that there is zero chance of no effect at all. This comparison could, of course, be reversed if EPA were to select one of the available statistically insignificant model results as its base case. However, that result also would be biased. The key point is that *any time a single base case model is adopted, the risk estimate that is produced using it will be biased.* Even if the base case model is selected such that its relative risk estimate lies near the middle of the range of all model results, its confidence interval will not be wide enough to reflect the true range of modeling uncertainty.

Another important point that emerges from this illustrative integrated uncertainty analysis is that *the true uncertainty is highly asymmetric* around EPA's base case point estimates. That is, the true probability distribution has much greater probability of risks below EPA's base case estimate than it has probability of risks above EPA's base case estimate. This directly contradicts the implication of EPA's "confidence intervals" which imply that the chances that risks are higher or lower than the point estimate are equal (*i.e.,* they follow the normal distribution associated with variances of statistically-estimated relative risks).

This asymmetry is because many of the modeling uncertainties that are ignored in EPA's base case approach would reduce the estimated risks resulting from that simplistic approach. These include: (a) possibilities of thresholds that the models have not been able to identify and (b) model results that are lower than the ones that EPA has selected. To the extent that the models not selected for the base case are statistically insignificant, a larger and larger pulse of probability becomes associated with the "no effect" outcome when these model results are incorporated into an integrated uncertainty analysis. This overstatement of risks from EPA's deterministic risk analysis approach was recognized by CASAC in its comments to EPA on the draft Staff Paper:

> "It is unfortunate that a more comprehensive, quantitative characterization of uncertainty has not been undertaken, even if it only took into account several sources of uncertainty simultaneously. ...There is also likely to be directionality to the degree of uncertainty, with greater uncertainty around effects at lower, compared with higher PM levels. Overall, the chapter tends to understate uncertainty, both through style, (*e.g.*, inclusion of numerically specific estimates, *e.g.,* "403" deaths rather than "400" or "about 400", and by not bringing together the individual sensitivity analyses."[23]

[22] The difference is due to higher as-is pollution levels in Los Angeles, as the same set of relative risk coefficients and weights are applied identically in both cities for long-term mortality risks.
[23] US EPA (2005), p. C-9.

One benefit of a more explicit, probabilistic approach is therefore that it provides a more complete and unbiased summary of the overall evidence than can be obtained with EPA's deterministic approach. Another benefit of an integrated uncertainty analysis, which is not possible to demonstrate with just these figures for as-is risk, is that an integrated uncertainty analysis can provide direct evidence of how the uncertainty in further health benefits starts to expand as one has to decide among lower and lower potential NAAQS levels. This concept of expanding uncertainty appears to be a rationale underlying some of EPA's discussions of where to set the $PM_{2.5}$ standards. It has obvious merits in a situation where the standard cannot be "risk-free," yet also cannot be set by balancing incremental costs against the incremental risk reduction. A natural step would be for EPA to adopt a probabilistic risk methodology that can directly provide estimates of the range of uncertainty in incremental risk reductions associated with incremental tightening of the standard.

III. EPA's "Evidence-Based" Approach Does Not Support Tightening the Daily Standard to 35 μg/m^3.

EPA makes a case to tighten the daily standard based, in part, on a risk analysis that overstates current evidence of $PM_{2.5}$ risks, and an incomplete summary of the trends in the risk analysis' estimates. In Part II of these comments, I presented my reasons for why that case to tighten the $PM_{2.5}$ NAAQS is not supported by the full evidence. However, even if one accepts that the daily standard should be tightened, EPA then uses an evidence-based approach in the PR to argue that the right level for a revised daily standard is 35 μg/m^3. I have reviewed this evidence-based argument, which appears on p. 2649 of the PR, and have a number of concerns with its technical basis. My concerns include: (a) that several of EPA's statements about the findings in specific epidemiological studies are incorrect or overstated, (b) that the PR offers an incomplete review of the full relevant body of evidence, also resulting in overstatement, and (c) that the presentation of evidence is so unclear that readers cannot readily observe that EPA's conclusions are not supported even by the evidence that the PR provides.

Corrections to Statements in PR's Evidence-Based Case

The case EPA makes for a daily $PM_{2.5}$ standard of 35 μg/m^3 on p. 2649 is strictly verbal, with very complex and lengthy sentences. While this is a confusing way to present any evidence, it also contains several factual misstatements. To start my review of the case, I have therefore reproduced the PR's exact text of this case in column 1 of Table 2, broken into its structural segments on separate, sequential rows of the table.[24] In column 2, I have then provided annotated comments on each of the PR's statements.

EPA's overall argument to set the daily $PM_{2.5}$ standard at 35 μg/m^3 is based on three parts:

(i) That there is much evidence of an effect in studies with 98th percentile values above 35 μg/m^3 (*i.e.*, in a range down to 39 μg/m^3),
(ii) That there is more mixed evidence among studies with 98th percentile $PM_{2.5}$ in the range of 30-35 μg/m^3,[25] and
(iii) That not much information is available for studies that had $PM_{2.5}$ 98th percentiles below 30 μg/m^3.

Following this structure, I have broken Table 2 into parts (i), (ii), and (iii).

[24] If one starts to read down column 1 from Part (i) to Part (ii) to Part (iii) of Table 2, one will have the entire text from p. 2649 of the PR's evidence-based argument for where to set the daily standard based on daily health effects studies for PM2.5. The only changes I have made are some added punctuation and formatting to enhance clarity.

[25] The gap between 39 μg/m^3 and 35 μg/m^3 is caused by a lack of any studies in this range. EPA could just as easily have defined the first range as being "down to 35 μg/m^3", or the second range as being "from 30-39 μg/m^3" without any loss of generality in its argument for setting the standard at 35 μg/m^3.

Table 2. Comments on Statements Made by EPA's in its Evidence-Based Case for a Daily $PM_{2.5}$ Standard of 35 $\mu g/m^3$.

Part (i): Statements about Studies with 98th Percentile Values Down to 39 $\mu g/m^3$

Based on the information in the Staff Paper and a supporting staff memo,[42] the Administrator observes an overall pattern of statistically significant associations reported in studies of short-term exposure to $PM_{2.5}$ across a wide range of 98th percentile values. More specifically, there is a strong predominance of studies with 98th percentile values down to about 39 $\mu g/m^3$ (in Burnett and Goldberg, 2003) reporting statistically significant associations with mortality, hospital admissions, and respiratory symptoms.	Burnett and Goldberg (2003) is a mortality paper for 8 Canadian cities. The 39 $\mu g/m^3$ is the 98thile provided by the authors to EPA for all 8 cities and, according to Ross and Langstaff (2005), is based on "averaged annual values for years in study" – there were 11 years. The act of averaging the values across the years will reduce the variance, and thus will generate a 98th percentile that could be substantially lower than the actual 98th percentile of the air concentrations in the dataset. Also, no information is provided on a city by city basis, so it is unclear what the 98th percentile was for the individual cities in that dataset. It is also unknown if only a couple of cities were driving the significant finding for the pooled set of cities. Thus, it seems inappropriate to use this particular study's 98th percentile estimate for attempting to identify boundaries where significance levels drop off. If this data point is dropped because of its variance-reducing bias, the next lowest 98th percentile value in this range would be 42 $\mu g/m^3$ associated with Schwartz (2003a) and Klemm and Mason (2003) – *i.e.*, for Boston in the "Six-Cities" study.
[42] As discussed in the Staff Paper (EPA, 2005a, p. 5–30) and supporting staff memo (Ross and Langstaff, 2005), staff focused on U.S. and Canadian short-term exposure $PM_{2.5}$ studies that had been reanalyzed as appropriate to address statistical modeling issues and considered the extent to which the reported associations are robust to co-pollutant confounding and alternative modeling approaches and the extent to which the studies used relatively reliable air quality data.	As will be noted below, several of the papers the PR cites in this excerpt are not mentioned on p.5-30 of the Staff Paper, nor in the supporting memo, Ross and Langstaff (2005). Further, several of the papers cited have not been reanalyzed to address statistical modeling issues (*i.e.*, the GAM problem). Also, as will be discussed below, most of the statistically significant associations found in the studies cited above were *not* robust to co-pollutant modeling, but only occurred in 1-P formulations. (Part IV of my comments also provides a detailed review of the lack of robustness of $PM_{2.5}$ associations to inclusion of gaseous co-pollutants, demonstrating the incorrectness of the statement in this footnote of the PR.)
For example, within this range of air quality, statistically significant associations were reported <u>for mortality</u> in:	
the combined Six City study (and three of the individual cities within that study) (Klemm and Mason, 2003),	As in the comment regarding Burnett and Goldberg (2003), there are difficulties using a 98th percentile for a group of cities, and so reference to the "combined Six City" results should be eliminated. <u>Four</u> of the six cities in the Six City study have 98th percentiles at or above 42 $\mu g/m^3$, and only one of those four cities had a statistically significant effect in a majority of the model formulations reported (*i.e.*, Boston – the city with the lowest 98th percentile of the group, at 42 $\mu g/m^3$). A second city (St. Louis) had mixed statistical significance. The remaining 2 cities (Steubenville and Knoxville) had no statistically significant results. Thus, although this study does report some statistically significant associations in the range down to 42 $\mu g/m^3$, its findings are far more mixed than EPA's statement suggests. This study only had 1-P formulations, leaving no indication of whether they might be robust to co-pollutant modeling.
the Canadian 8-City Study (Burnett and Goldberg, 2003),	This study does present robustly statistically significant associations for the combined set of eight cities. However, it does not reveal any city-specific results, to help understand if the associations are related to all or just a few of the 8 cities. Further, it is very difficult to know how to characterize a 98th percentile that can be associated with these findings. The level of 39 $\mu g/m^3$ is biased downwards by an unknown degree.
and in studies in Santa Clara County, CA (Fairley, 2003)	This study reports associations that are statistically significant for same-day $PM_{2.5}$, but which is <u>negative</u> for $PM_{2.5}$ at a one-day lag. Only the former was was subjected to co-pollutant modeling, and it was robustly significant. The 98th percentile for this dataset is 59 $\mu g/m^3$

and Philadelphia (Lipfert *et al.*, 2000a);	This study's results were not at all robust to 2-P formulations, and it does not support a statement in favor of a "strong predominance" of statistical significance in this range of air quality. In fact, the authors concluded that the associations observed in 1-P formulations should actually be attributable to ozone, not $PM_{2.5}$, based on their response to co-pollutant modeling.
for hospital admissions and emergency department visits:	
in Seattle (Sheppard, 2003),	The statistical significance found in this paper is limited to a 1-P model formulation using the GAM estimation technique. GLM and 2-P formulations are also reported in this paper, and they are a mix of borderline significant and insignificant.
Toronto (Burnett *et al.*, 1997; Thurston *et al.*, 1994),	Burnett *et al.* (1997) use the GAM method and has not been reanalyzed. Its inclusion here violates EPA's policy not to rely on such studies unless they have been reanalyzed. Even so, it did not find robustly significant associations. Thurston *et al.* (1994) also did not find robustly significant associations for $PM_{2.5}$, especially after considering the role of ozone in co-pollutant modeling. They state at p.282: "This points out the importance of considering as many pollutants as possible in such analyses, in order to diminish the chances of being misled as to which of the many ambient air pollutants is actually culpable for any noted air pollution-health effects associations."
Detroit (Ito, 2003, for ischemic heart disease and pneumonia, but not for other causes),	Ito (2003) considers 4 types of cardiac admissions and 2 types of respiratory admissions in 1-P formulations only. He does find a statistically significant association for pneumonia, and a borderline significant association for heart failure. Ischemic heart disease was *not* statistically significant, contrary to EPA's statement; nor were three other categories considered. No gaseous co-pollutant modeling results were reported.
and Montreal (Delfino *et al.*, 1998, 1997, for some but not all age groups and years);	Delfino (1998) finds only a borderline significant $PM_{2.5}$ association in a 1-P formulation that disappears in a 2-P formulation, with ozone becoming the dominant explanatory pollutant. [26] Delfino (1997) has a 98th percentile of 31.2 $\mu g/m^3$and thus should not be placed in this group of studies for a range of "down to 39 ug/m3). Furthermore, its findings are more mixed than this suggests. It does find a statistically significant association between $PM_{2.5}$ and emergency room visits for >64 years age in a 1-P formulation, but its significance is utterly eliminated in a 2-P formulation (where ozone, however, remains significant).
for respiratory symptoms in panel studies:	
in a combined Six City study (Schwartz *et al.*, 1994)	Schwartz *et al.* (1994) uses the GAM method and has not been reanalyzed. Its inclusion here violates EPA's policy not to rely on such studies unless they have been reanalyzed. Additionally, this study is for a combined set of cities, without reporting city-specific associations or city-specific 98th percentile values. Therefore, as is the case with Burnett and Goldberg (2003), it is difficult to use the 98th percentile value reported for this study to attempt to determine where to set a daily standard. The 98th percentile value is not provided in the supporting staff memo that the PR cites. A memo released only on April 5, 2006 reveals its 98th percentile level is 48$\mu g/m^3$.
and in two Pennsylvania cities (Uniontown in Neas *et al.*, 1995; State College in Neas *et al.*, 1996);	These 2 papers also are not mentioned in the supporting staff memo that the PR cites. A memo released only on April 5, 2006 (Ross and Langstaff (2006)) reveals these datasets have 98th percentile levels of 60 and 69 $\mu g/m^3$, respectively. Despite having relatively high PM levels compared to other studies that EPA has considered, both papers find

[26] The PR actually cites to the wrong Delfino *et al.* (1998) paper, referencing a paper about PM_{10} and asthma in Los Angeles. I use the correct citation in my comments here.

	mixed results over 4 types of respiratory symptoms (colds, cough, wheeze, and changes in PEFR). Across both studies, only cough was associated with $PM_{2.5}$ in a statistically significant manner under both 1-P and 2-P formulations.
and for lung function in Philadelphia (Neas *et al.*, 1999).[43]	None of the associations between PEFR and $PM_{2.5}$ in this paper are statistically significant, even in 1-P modeling. (EPA's statement on p. 2630 of the PR that associations in this paper are statistically significant is not supported by a review of the original paper.) Again, this paper is not mentioned in the supporting staff memo that the PR cites. Ross and Langstaff (2006)) reveals this dataset has a 98th percentile levels of 45 $\mu g/m^3$.
[43] Of the studies within this group that evaluated multipollutant associations, as discussed above in section II.A.3, the results reported in Fairley (2003), Sheppard (2003), and Ito (2003) were generally robust to inclusion of gaseous co-pollutants, whereas the effect estimate in Thurston *et al.* (1994) was substantially reduced with the inclusion of O_3.	Ito (2003) did not present any 2-P formulations with gaseous co-pollutants included. The only 2-P formulations shown included coarse fraction simultaneously with $PM_{2.5}$, and in those results, even the few significant associations (pneumonia and heart failure hospital admissions) became insignificant. Part IV of my comments provides a detailed review of the evidence on inclusion of gaseous co-pollutants, and demonstrated that the results are not "generally robust" as this footnote of the PR says.
Studies in this air quality range that reported positive but not statistically significant associations with mortality include studies in:	As the entire logic of this analysis is to seek a level of air quality at which the degree of statistical significance in findings starts to drop off, studies that find positive associations that do not rise to the level of statistical significance should be viewed as examples that undermine the statement that there is a predominance of statistically significant results within this range has been extended too low. For example, Schwartz and Neas (2000) state that "lower respiratory symptoms in a two-pollutant model were associated with …fine particles…but not coarse particles" and the supporting evidence for this statement was that there was an odds ratio >1 for $PM_{2.5}$ that was statistically significant, and there was an odds ratio >1 for coarse particles also, but it was *not* statistically significant (Schwartz and Neas, 2000, p. 6).
Detroit (Ito, 2003),	This study found no statistically significant association between $PM_{2.5}$ and mortality at a 98th percentile of 55 $\mu g/m^3$.
Pittsburgh (Chock *et al.*, 2000),	This study found no statistically significant association between $PM_{2.5}$ and mortality at a 98th percentile of about 75 $\mu g/m^3$.
and Montreal (Goldberg and Burnett, 2003).	This study found no statistically significant association between $PM_{2.5}$ and mortality at a 98th percentile of about 53 $\mu g/m^3$.

Part (ii): Statements about Studies with 98th Percentile Concentrations between 30 and 35 $\mu g/m^3$

Within the range of 98th percentile $PM_{2.5}$ concentrations of about 35 to 30 $\mu g/m^3$, this strong predominance of statistically significant results is no longer observed. Rather, within this range,	
some studies report statistically significant results:	The two examples cited next should not be characterized as reporting statistically significant "results", but only as having some model formulations that are statistically significant. Neither of the two papers cited rise to the level of having found an *overall* statistically significant set of associations, as explained below, but even these two papers individually report mixed results, at best. The main distinction in the evidence cited in this clause and the following clause is that Mar *et al.* and Ostro *et al.* are *mortality studies,* while Delfino *et al.* and Peters *et al.* are

	morbidity studies -- but all four of them find "mixed results."
Mar *et al.*, 2003;	Mar *et al.* did report some statistically significant results, but the statistical significance was not robust in about 2/3 of their results. The data for Mar *et al.* (2003) was for Phoenix, and was studied by three different sets of researchers, including also Smith *et al.* (2000) and Clyde *et al.* (2000). The findings for Phoenix should reflect this entire suite of modeling using the same data, and those results are far more mixed than represented by Mar *et al.* alone.
Ostro *et al.*, 2003.	This study finds only one statistically significant result, while the majority of the associations it reports between $PM_{2.5}$ and mortality are insignificant.
other studies report mixed results in which some associations reported in the study are statistically significant and others are not:	
Delfino *et al.*, 1997	This study finds a statistically significant association between $PM_{2.5}$ and emergency room visits for >64 years age in a 1-P formulation, but its significance is utterly eliminated in a 2-P formulation (where ozone, however, remains significant). Other associations reported were not significant even in a 1-P formulation.
Peters *et al.*, 2000	This was a study of the frequency of discharge of implanted defibrillators, effectively a "symptoms" study. For 6 of 100 patients in the study, one out of several $PM_{2.5}$ associations (all 1-P) was significant, but "the strongest associations were observed for NO_2....including both pollutants into one model reduced the effect estimate of $PM_{2.5}$ effectively to 0, whereas the effect estimate of NO_2 was unchanged." There was no statistically significant association for $PM_{2.5}$ and defibrillator discharges in any of the other 100 patients in the study. This paper also was not cited in Ross and Langstaff (2005). Ross and Langstaff (2006), released only on April 5, 2006, shows its 98th percentile to be 31.7 $\mu g/m^3$.
and another study reports associations in two of six cities that are not statistically significant (Klemm and Mason, 2003).[44]	This clause moves back to a *mortality* study. It is misleadingly worded, and needs clarification: Within the six cities studied in Klemm and Mason (2003), there are two cities whose 98th percentiles are in the range of 30-35 $\mu g/m^3$, and *neither* has any significant findings even in the 1-P formulations that are all that are presented. (The other four cities are in the range of >39 $\mu g/m^3$, they are mostly insignificant as well.)
[44] For example, Delfino *et al.* (1997) report statistically significant associations between $PM_{2.5}$ and respiratory emergency department visits for elderly people (>64 years old), but not children (<2 years old) in one part of the study period (summer 1993) but not the other (summer 1992). Peters *et al.* (2000) report new findings of associations between fine particles and cardiac arrhythmia, but the Criteria Document observes that the strongest associations were reported for a small subset of the study population that had experienced 10 or more defibrillator discharges (EPA, 2004, p. 8--164).	This footnote is consistent with my comments above. It should have been placed with the preceding clause.

Part (iii): Statements about Studies with 98th Percentile Concentrations below 30 μg/m³

Further, the very limited number of studies in which the 98th percentile values are below this range [*i.e.*, 30 to 35 μg/m³] do not provide a basis for reaching conclusions about associations at such levels:	
Stieb *et al.*, 2000	Stieb *et al.* (2000) used the GAM method and it has not been reanalyzed. Its inclusion here violates EPA's policy not to rely on such studies unless they have been reanalyzed. It should be dropped, leaving only one study in this range at all.
Peters *et al.*, 2001	This study reports a significant association between myocardial infarction and $PM_{2.5}$ in 1-P formulations. No 2-P formulations were presented. However, the study uses a procedure for controlling for $PM_{2.5}$ that has since been shown to be biased (Jane, Sheppard & Lumley, 2004). This paper also was not cited in Ross and Langstaff (2005). Ross and Langstaff (2006), released only on April 5, 2006, shows its 98th percentile to be 28.2 μg/m³.

More Complete Information to Supplement Evidence-Based Case in PR

After making the corrections to the factual inaccuracies that I have identified in column 2 of Table 2, the evidence of a "strong predominance" of significant associations for dataset with 98th percentiles above 39 $\mu g/m^3$ appears to be unsupported. Additionally, the dividing line at 39 $\mu g/m^3$ is not a location where one starts to find clearly less mixed results above than below. Even so, the evidence cited in PR is not complete.

In order to perform a more complete review of the body of evidence on $PM_{2.5}$, I developed a list of all the epidemiological studies for short-term $PM_{2.5}$ that I could identify that met the following criteria:

- Is cited in the CD (papers published after the CD cut-off date are therefore not included, since these are not supposed to be a part of EPA's current evidence-based rationale).
- Is a short-term health effects study.
- Is based on US or Canadian datasets.
- Has no GAM problem. (If a GAM problem existed in a paper, only reanalyzed results were considered.)[27]
- Used directly measured $PM_{2.5}$. (Studies that "filled" missing values were included, but studies that estimated all the $PM_{2.5}$ values from visibility or other measures were not included.)
- Considered any type of effect that could be categorized as a clear health impact. This included aggravation of asthma, changes in lung function measures (*e.g.*, PEFR, FEV1), and detected arrhythmias.[28]

I found studies for 38 specific combinations of type of health effects and $PM_{2.5}$ dataset, that I call "locations," and list in Table 3 . I report cities' results individually wherever possible, if the study is a multi-city study. Two multi-city papers do not provide city-specific data: Burnett and Goldberg (2003) and Schwartz and Neas (2000). Of these 38 "locations" 25 are cited on p. 2649 of the PR. I reviewed each study to determine the general significance level that it found for the $PM_{2.5}$ association specifically. I ranked them into one of three categories: "no overall significant association," "mixed significance" and "overall significant association." By "overall significant association," I mean that a majority of the regressions in the paper produced statistically significant associations. If a 2-P result is provided, it must also be statistically significant to be placed in this category, unless there is evidence of

[27] This requirement caused me to drop three of the studies that the PR cites in its case on p. 2649: Stieb *et al.* (2000), Burnett *et al.* (1997), and Schwartz *et al.* (1994). However, the last of these was replaced in my review by Schwartz and Neas (2000) which analyzes the same effects, and finds the same general associations for $PM_{2.5}$, but which does not appear to have the kind of GAM usage that was subject to the convergence problem.

[28] It did not include measures of heart rate variability, an association with which may be indicative of some kind of physiological response to $PM_{2.5}$ exposure, but whose significance to actual health outcomes remains unknown. This criterion only excluded two studies that otherwise fit these criteria: Gold (2003) and Liao *et al.* (1999), neither of which EPA uses in its evidence-based case for setting the standard at 35 $\mu g/m^3$.

multicollinearity problems in the 2-P model.[29] A ranking of "no overall significant association" was assigned if the majority of the results in the paper are insignificant even if a statistically significant result exists in the paper. If there is only one 1-P and one 2-P result reported, and the 2-P is insignificant, I assigned it to this category, unless there is evidence of multicollinearity problem in that the 2-P result.

My specific rankings are shown in Table 3. Appendix A provides my rationale for each assigned ranking. Table 3 also shows the 98th percentile $PM_{2.5}$ level associated with each location. Where possible, these values are from Ross and Langstaff (2005) or Ross and Langstaff (2006). However, many of these studies are not listed in those documents, and I estimated their 98th percentile $PM_{2.5}$ from other relevant the distributional data provided in the cited paper(s). My estimation methods are documented in Appendix B. However, it should be noted that all the values that I had to estimate fall above the 40 $\mu g/m^3$ level, and so my estimates do not affect how one might consider setting a standard in ranges from 40 $\mu g/m^3$ downwards, if that is the interval of concern.

[29] Such evidence exists when both the PM and gaseous pollutant would become insignificant in a 2-P formulation even though both are significant in their respective 1-P formulations.

Table 3. List of Locations and Associated Papers Reporting Short-Term Epidemiological Findings

Location	Impact Category	Papers Studying this Impact for this Location	98th Percentile $PM_{2.5}$	Robustness of Association for $PM_{2.5}$ (*)
Newark, NJ	Mortality	Tsai *et al.* (2000)	94.2	2
Camden, NJ	Mortality	Tsai *et al.* (2000)	84.4	2
Elizabeth, NJ	Mortality	Tsai *et al.* (2000)	84.0	1
Steubenville, OH	Mortality	Schwartz (2003a), Klemm and Mason (2003)	81.5	2
Los Angeles, CA	Mortality	Moolgavkar (2003)	60.4	1
Santa Clara County, CA	Mortality	Fairley (2003)	59.0	3
Pittsburgh, PA	Mortality	Chock *et al.* (2000)	56.3	1
Detroit, MI	Mortality	Ito (2003), Lippmann *et al.* (2000)	55.2	1
Montreal, Canada	Mortality	Goldberg and Burnett (2003)	53.1	1
Philadelphia, PA	Mortality	Lipfert *et al.* (2000a)	44.2	2
St. Louis, MO	Mortality	Schwartz (2003a), Klemm and Mason (2003)	43.6	2
Knoxville, TN	Mortality	Schwartz (2003a), Klemm and Mason (2003)	43.5	1
Boston, MA	Mortality	Schwartz (2003a), Klemm and Mason (2003)	42.0	3
8 Canadian Cities	Mortality	Burnett and Goldberg (2003)	38.9	3
Madison, WI	Mortality	Schwartz (2003a), Klemm and Mason (2003)	34.3	1
Coachella Valley, CA	Mortality	Ostro *et al.* (2003)	33.4	1
Phoenix, AZ	Mortality	Mar *et al.* (2003), Smith *et al.* (2000) and Clyde *et al.* (2000)	32.2	2
Topeka, KS	Mortality	Schwartz (2003a), Klemm and Mason (2003)	32.0	1
Los Angeles, CA	Morbidity: Hospital Visits	Moolgavkar (2003)	60.4	1
Detroit, MI	Morbidity: Hospital Visits	Ito (2003)	55.2	3
Toronto, Canada	Morbidity: Hospital Visits	Thurston *et al.* (1994)	51.0	1
Seattle, WA	Morbidity: Hospital Visits	Sheppard (2003)	46.6	2
Atlanta, GA	Morbidity: Hospital Visits	Tolbert *et al.* (2000)	41.5	1
Montreal, Canada	Morbidity: Hospital Visits	Delfino *et al.* (1998)	40.7	1
Montreal, Canada	Morbidity: Hospital Visits	Delfino *et al.* (1997)	31.2	1
Boston, MA	Morbidity: Hospital Visits	Peters *et al.* (2001)	28.2	3
Los Angeles, CA	Morbidity: Symptoms	Ostro *et al.* (2001)	112.0	3
State College, PA	Morbidity: Symptoms	Neas *et al.* (1996), Schwartz and Neas (2000)	69.0	2
Denver, CO	Morbidity: Symptoms	Ostro *et al.* (1991)	60.3	1
Uniontown, PA	Morbidity: Symptoms	Neas *et al.* (1995), Schwartz and Neas (2000)	60.0	3
Los Angeles, CA	Morbidity: Symptoms	Linn *et al.* (1999)	59.1	1
San Diego, CA	Morbidity: Symptoms	Delfino *et al.* (1996)	51.1	1
6 US Cities	Morbidity: Symptoms	Schwartz and Neas (2000)	48.0	3
Virginia	Morbidity: Symptoms	Naeher *et al.* (1999)	45.1	2
Virginia	Morbidity: Symptoms	Zhang *et al.* (2000)	45.1	1
Philadelphia, PA	Morbidity: Symptoms	Neas *et al.* (1999)	44.9	1
New Hampshire	Morbidity: Symptoms	Korrick *et al.* (1998)	41.2	2
Massachusetts	Morbidity: Symptoms	Peters *et al.* (2000)	31.7	1

(*) 1="no overall significant association," 2="mixed significance of findings" and 3="overall significant association." See Appendix A for my definition of criteria for these three categories and my rationale for assigned rankings.

Synthesizing and Interpreting the Full Body of Evidence

Information as extensive and complex as that in Table 3 is easier to interpret if presented in alternative formats. Figure 7 presents a graphical summary for all the relevant studies that EPA should use in making its evidence-based case, based on the data in Table 3. The blue diamonds are studies EPA cited on p. 2649, and the red diamonds are studies EPA has not called (and for which I therefore had to estimate the 98th percentile value). The Burnett and Goldberg (2003) and the Schwartz and Neas (2000) studies are shown as an unfilled diamonds to emphasize that these 98th percentile values are for a combination of individual cities, and therefore cannot be compared to the others in this analysis. The two dotted red horizontal lines reflect the dividing lines of the three categories in EPA's evidence-based argument.

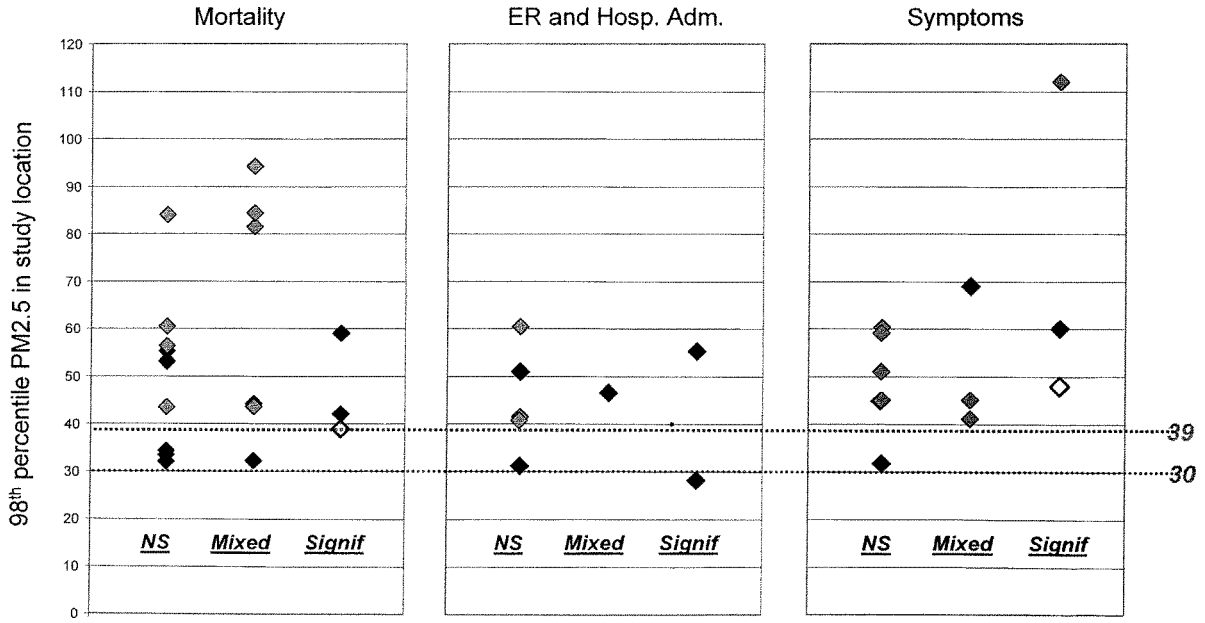
When this complete set of evidence is organized into this internally-consistent summary format, it becomes clear that EPA's arguments to set the standard at 35 $\mu g/m^3$ do not conform with the evidence. For example, there is no evidence of a "predominance of statistically significant findings" above the 39 $\mu g/m^3$ line. Nor is there any higher level of $PM_{2.5}$ 98th percentile above which statistical significance is more common than below. While it is true that evidence of significance is mixed for studies in the 30-35 $\mu g/m^3$ range, it is just as mixed for studies above the current standard of 65 $\mu g/m^3$ – and particularly so for the category of mortality.

When the Burnett and Goldberg study (the unfilled diamond) is ignored for the reasons stated above, the lowest 98th percentile level for which a statistically significant mortality effect is robust is at 42 $\mu g/m^3$ – *and this is for Boston from the Six Cities database on which the current standards were based in the first place.* In other words, if considering just the mortality evidence, EPA is left with making a case to tighten the daily standard using the same study that was used when the present standards were set. Moreover, since that time, the overall robustness of the association in that study has been proven to be less than it was thought to be in 1997. Further, among the mortality studies, only 2 out of 10 that are in the range of 42 $\mu g/m^3$ up to the current daily standard of 65 $\mu g/m^3$ find robust statistical significance. The second of these is Fairley (2003) for San Jose, with a 98th percentile of 59 $\mu g/m^3$.

Turning to the morbidity evidence, little further guidance appears. For hospital admissions and emergency room visits, there is no pattern of significance at all. There is one study at the very low 98th percentile of 28.2 $\mu g/m^3$ that finds a robustly significant result, which is Peters *et al.* (2001). The only other study in this category with a robustly significant effect has a 98th percentile of 55.2 $\mu g/m^3$ (*i.e.*, Ito (2003)), and there are five studies in the intervening $PM_{2.5}$ levels that find do not find a robust association.

Figure 7. Summary of Robustness of Statistical Significance as Function of $PM_{2.5}$ 98th Percentile, for Three Categories of Severity of Health Effects.

Blue diamonds are for studies where EPA has directly reported 98th percentile values in the study. Red diamonds are for studies that EPA has not included in its evidence-based arguments and has not provided 98th percentile information. For these studies, 98th percentile $PM_{2.5}$ has been estimated from other relevant data in the respective publications, as documented in Appendix B. Unfilled diamonds are for studies where multiple cities' data were combined, thus not reflecting a true population-level 98th percentile exposure.
"NS"=no overall significance in dataset"; "Mixed"=mixed significance of findings for the dataset; "Signif"=overall significant associations in the dataset.



The third category is studies of respiratory and cardiovascular symptoms. These studies mostly consider changes in measures of pulmonary function (*e.g.*, PEFR and FEV1), or in reported incidence of coughing or wheezing. One study in this group assessed changes in frequency of cardiac arrhythmia, using defibrillator data (it did not find a robust significant effect however.) Once again, there are too few studies with robustly significant findings to discern any trend.

The evidence thus does not provide a coherent case for setting the daily standard at 35 $\mu g/m^3$. It also does not provide a case for tightening the daily standard, given the continued prominence of the Boston mortality association within the statistically significant results. And most of all, the evidence does not provide any apparent alternative level at which to set the standard, if one is intent on tightening it.

Nevertheless, there are some studies that do find robust associations at levels below the current standard of 65 $\mu g/m^3$. Thus, if one is intent on tightening the daily $PM_{2.5}$ standard, one's best approach might be to start by exploring the methodological merits of individual studies finding robust associations at the lowest $PM_{2.5}$ levels, and work upwards until a study is found that has strong methodological properties. I will therefore go through this exercise next.

The study with robust significant findings at the lowest $PM_{2.5}$ 98^{th} percentile level is Peters *et al.* (2001), followed by Burnett and Goldberg (2003), then the Boston results from the Six Cities database.

Peters *et al.* (2001). This paper is the one with the lowest 98^{th} percentile of all those identified, yet it also is among the few that report a robustly significant association between $PM_{2.5}$ and health – in this case, with likelihood of onset of myocardial infarction. The 98^{th} percentile in the time period studied was 28.2 $\mu g/m^3$. The method used is different from all of the other short-term $PM_{2.5}$ mortality and morbidity papers reviewed here – a "case-crossover approach." Most short-term health effects studies for mortality and hospital visits/admissions explore the association between daily numbers of deaths or hospital visits/admissions and daily air quality. Individuals are not tracked at all. The case-crossover method is quite different in that it identifies individuals who have experienced a particular health event (myocardial infarction, in this case), and then explores the differences between air quality just prior to the time of that event and at other, "referent times" when the event did not occur.

The case-crossover design is an accepted statistical approach, considered to have substantial merits for use in epidemiology. However, it is also a relatively new approach and its use in Peters *et al.* (2001) stands as a methodological outlier within the $PM_{2.5}$ epidemiological literature reflected in Table 3 and Figure 7 above. Given the fact that this paper also appears to provide the strongest case for possibly tightening the $PM_{2.5}$ daily standard, its application of this relatively new statistical approach merits some scrutiny.

A critical issue in case-crossover design is how to select the "referent times" for the statistical controls. This is a judgment that is in the hands of the researcher and there are many alternative ways that the referent times can be selected – each of which can produce different

statistical findings, of course. Some referent selection methods can introduce statistical biases, and thus are less desirable. Janes *et al.* (2004) characterize the biases associated with alternative referent selection schemes, and report that a "restricted unidirectional" selection method is subject to intractable forms of biases. Peters *et al.* (2001) use this restricted unidirectional method. Of nineteen case-crossover studies of air pollution exposures between 1999 and 2004 noted by Janes *et al.*, Peters *et al.* (2001) appears to be the only one that used this biased referent selection method exclusively.

In light these statistical concerns with the way Peters *et al.* (2001) have applied the case-crossover approach, it would be prudent not to let this single study serve as a basis for where to set the $PM_{2.5}$ daily standard. Additionally, this study did not report any $PM_{2.5}$ results that also controlled for gaseous pollutants. Part IV of my comments explains why this should be another cause for caution in the weight that this study should receive in an evidence-based approach.

Burnett and Goldberg, 2003. This paper is a reanalysis of part of a much more extensive study, Burnett *et al.* (2000), that is affected by the GAM default setting problem. This is a study of short-term mortality in eight cities in Canada. Across a range of 1-P formulations, this paper does find mostly statistically significant associations between mortality and $PM_{2.5}$. However, the only results reported are for all eight cities combined, yet alternative smoothing strategies reported in Burnett and Goldberg (2003) – particularly those that allowed the smoothing to be different for each city – substantially reduce the size of the $PM_{2.5}$ relative risk estimate and also render the $PM_{2.5}$ association statistically insignificant.

The authors note that there is insufficient evidence from this study to conclude that the association varies across the cities. Nevertheless, their results also make it difficult to consider using the 98th percentile *across all eight cities* as an indicator of what level of 98th percentile in a given city might account for the associations observed in this study. The 98th percentile of 38.9 $\mu g/m^3$ reported in Ross and Langstaff (2005) is for all eight cities combined. It is not clear how the city's individual daily $PM_{2.5}$ values were used to develop a single 98th percentile for the combined set, but air quality summary statistics in the original paper (Burnett *et al.* (2000)) indicate that the 98th percentiles for the eight individual cities likely range between about 27 and 48 $\mu g/m^3$. If the statistically significant association is being driven by one or more of the cities with higher levels of PM, then the 98th percentile that should be assigned to this study would be higher than 39 $\mu g/m^3$. (The opposite could be true as well, but if the associations are being driven by cities with lower rather than higher $PM_{2.5}$, this would raise yet other important questions for the NAAQS.)

Thus, a primary concern with relying on the data point of 39 $\mu g/m^3$ from this study to consider where to set a daily standard for $PM_{2.5}$ is the fact that this value is not comparable to the 98th percentiles for all of the other studies whose findings are being evaluated in this manner.[30] There are, however, a number of other issues associated with use of this study that merit mention.

[30] Note, for example, that my analysis breaks the results from the Six Cities studies of mortality into their individual city-specific 98th percentiles.

First, the original study did consider the role of $PM_{2.5}$ in conjunction with gaseous co-pollutants, and those analyses found that $PM_{2.5}$ and $PM_{10-2.5}$ together appeared to have much less explanatory power than the gaseous pollutants. These multi-pollutant explorations were not repeated for the reanalysis, so we cannot know if this result would remain when applying correct statistical methods. However, at present, the results EPA is using in its evidence-based approach are strictly from 1-P models. As Part IV of my comments will explain, that in itself is a concern. However, the concern is heightened in this case because earlier analyses suggested that indeed the $PM_{2.5}$ association in this study is *not* robust to inclusion of gaseous pollutants.

Second, Montreal is one of the eight cities in this study. The same core group of researchers (Goldberg and Burnett (2003)) could find no statistically significant associations for Montreal alone using similar methodology. Delfino *et al.* (1997 and 1998) were unable to find any morbidity associations for $PM_{2.5}$ in Montreal. Toronto is also one of the eight cities and Thurston *et al.* (1994) could find no $PM_{2.5}$ association with morbidity there. These other studies raise concerns about the role of individual cities in driving the combined-city associations reported in Burnett and Goldberg (2003), yet there is no such information available to better assess and understand the implications and robustness of its findings.

Boston results from Six Cities Dataset. Boston is one of the cities for which a statistically significant short-term $PM_{2.5}$ mortality association was first reported in Schwartz *et al.* (1996). The current $PM_{2.5}$ standards were based on the association found for Boston in this dataset. Although the 98th percentile for Boston in this dataset is 42 $\mu g/m^3$, the combination of an annual standard of 15 $\mu g/m^3$ and a daily standard of 65 $\mu g/m^3$ was found to provide the requisite level of public health protection in Boston and elsewhere.

Since 1997, the Boston results have been reanalyzed in Schwartz (2003a) and in Klemm and Mason (2003). The original GAM-based finding was not much affected by reanalysis with correct convergence settings. However, a series of alternative temporal smoothing, and linear estimation methods were explored. Alternative degrees of smoothing did produce a progressive decline in the original size of effect, and in the most controlled case, the $PM_{2.5}$ association was statistically insignificant.[31] Nevertheless, this Boston dataset provides one of the lowest 98th percentile levels for which a robust association appears.

A final note that has heightened relevance today compared to 1997 is that the only associations reported from the Boston dataset are single-pollutant formulations. As Part IV of my comments explains, there is substantial evidence available since 1997 to know that 1-P formulations generally overstate the role of $PM_{2.5}$.

Fairley (2003). This paper is often also cited as one of the reasons to lower the standard, but as can be seen, it actually has a relatively high 98th percentile of 59 $\mu g/m^3$. Nevertheless, its annual average $PM_{2.5}$ level (13.6 $\mu g/m^3$) is among the lowest of the studies with robustly significant associations, and a closer look at some of the features of this study also is warranted.

[31] The pattern of sensitivity in these extra analyses can be observed in Figure 3 above. That figure shows the combined-city result's sensitivity, but it is mirrored by the Boston city-specific results, which appear to be a key driver of the combined-city effects.

A key attribute of the dataset used for this study is the magnitude of the decline over time in $PM_{2.5}$ levels that occurs within the period analyzed. Table 4 shows that although the 98th percentile and annual average levels in this dataset were below the current standards *when averaged over the entire time period*, $PM_{2.5}$ levels were actually quite high in the earliest years of the study period. There is no discussion or information provided in the paper about the possibility that the $PM_{2.5}$ associations found in this dataset might be driven by the higher levels in earlier years, or how they vary over time. Few other epidemiological papers address this question, but few of them have relied on data with such pronounced trends in the air quality being associated with health effects.

Table 4. $PM_{2.5}$ Levels ($\mu g/m^3$) in Dataset Used in Fairley (2003)
Source: Fairley (1999)

	1990	1991	1992	1993	1994	1995	1996
98th percentile	88	51	48	50	44	32	25
Annual mean	18.4	15.5	13.8	12.9	12.6	10.3	9.5

One other concern associated with this study is the fact that the significant $PM_{2.5}$ association occurs for same-day $PM_{2.5}$, but the association is actually quite negative when a 1-day lag is considered. While a true causal association would likely reveal greater effects with some lags than with others, it does raise some concern when a large and robust effect for one lag is accompanied by a complete reversal of the association for another lag that differs by only a single day. The same-day $PM_{2.5}$ association is robust in various formulations including gaseous pollutants, but the negative association for a 1-day lag is never again explored.

In conclusion, there are a number of significant questions remaining regarding Fairley (2003) that makes it a poor candidate as a basis for a tighter daily $PM_{2.5}$ standard.

Conclusions from the More Complete Evidence-Based Approach

In this section, I have reviewed each of the statements made by EPA in its evidence-based case in the PR and made a number of corrections. I have also identified and incorporated elements of the relevant literature that are missing from the case presented in the PR. Finally, I summarized this information in a graphical format more useful for interpretation and decision making. Having done this, it became apparent that EPA's arguments for setting the daily standard at 35 $\mu g/m^3$ are not supported by the evidence. I find that there is in fact no obvious level above which there is a clear "predominance" of significant studies. As I have explained in Part II, there is no clear case in the risk analysis to tighten the daily standard at all. That finding is probably related to the problem of finding a reasonable level for a standard using an evidence-based approach. Nevertheless, a complete analysis of the current evidence leaves EPA with a quite arbitrary decision on where to draw the line.

With the choice of where to set the standard not possible to be guided by any patterns or trends in the evidence, one is forced to consider the individual merits of just a few key studies that are salient in that they do find statistically significant results at air quality levels below the current standards. My review in this manner leads me to conclude that Peters *et al.* (2001)

poses too many significant methodological concerns to be a basis for a stringent daily standard. The next lowest study, Burnett and Goldberg (2003), leaves some important methodological questions, but the primary difficulty in relying on this study to set a daily standard is that its reported 98th percentile level is not really a value associated with a single location that has been demonstrated to have a $PM_{2.5}$-health association. The relevant $PM_{2.5}$ level to associate with that study is likely above the combined-city level of 39 $\mu g/m^3$, perhaps in the mid to high 40s.

This brings us to the Boston dataset from the Six Cities study with a 98th percentile of 42 $\mu g/m^3$. The primary methodological concern with this study is that it relies on only a 1-P formulation (as do the other two discussed above). Ironically, this is the same dataset on which the current standards were based, and since that time, some additional uncertainties associated with its findings have been elucidated in reanalyses. Nevertheless, it remains a study that one could argue would still serve as a lower bound for setting a daily standard. Given that the current standards were set on the basis of this study originally, and the somewhat eroded robustness of its findings since then, one could reasonably conclude not to tighten the current standard at all. This would be consistent with my overall conclusions that the estimated risks are lower now than in 1997, and that uncertainties with the epidemiological evidence have become heightened since 1997.

IV. EPA Understates the Sensitivity of $PM_{2.5}$ Risk Associations to Inclusion of Gaseous Co-Pollutants

The PR repeatedly states that the $PM_{2.5}$ associations with health are "generally robust" to the inclusion of gaseous pollutants in a 2-P model formulation. This statement is offered as a reason that the evidence is stronger for these associations now than in 1997. It is also used as a reason to rely strictly on 1-P model results in the risk analysis, even when 2-P results are available. Much therefore rests on this statement, yet it is inconsistent with the actual evidence. In this part of my comments, I document how the inclusion of gaseous co-pollutants in a statistical model generally *erodes* the confidence in any $PM_{2.5}$ association that might be supported by a 1-P model result.

Short-Term Studies

Following the selection criteria I described in Part III for identifying short-term $PM_{2.5}$ health effects studies, I identified 34 papers that provided estimates of the association between $PM_{2.5}$ and one or more health endpoints, ranging from mortality to subtle changes of unknown significance, such as heart rate variability (HRV). Of the 34 papers, 11 reported both 1-P results for $PM_{2.5}$ and also 2-P results that included at least one gaseous co-pollutant simultaneously with $PM_{2.5}$.[32] Table 5 lists those papers and summarizes the outcomes of the 1-P and 2-P formulations. All but one of those studies did find a statistically significant $PM_{2.5}$ association in a 1-P formulation. Table 5 shows that in all but two of those cases where the 1-P formulation found a significant association with $PM_{2.5}$, the $PM_{2.5}$ association became insignificant in the 2-P model. Additionally, in all but one of *those* cases, the gaseous co-pollutant would remain significant, thus eliminating an argument that the sensitivity of the $PM_{2.5}$ association must be due to multicollinearity.[33] (If multicollinearity were a problem, both pollutants would become insignificant.)

Of the 11 studies that included both 1-P and 2-P results, only two studies (Fairley (2003) for mortality and Gold (2003) for heart rate variability) found a $PM_{2.5}$ effect that was robust to inclusion of gaseous pollutants. This is quite strong evidence that EPA is incorrectly stating in the PR that 1-P formulations are reasonable to continue to use. This is an important point because EPA is relying primarily on 1-P results to build its case for the need to tighten the $PM_{2.5}$ standards, both in the quantitative risk analysis and in its evidence-based approach.

Table 6 lists the 22 papers that report only 1-P model results. A majority of them are being used in the PR as part of the evidence in favor of tightening the daily $PM_{2.5}$ standard.

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[32] A 12th paper (Zhang et al. 2000) also performed multi-pollutant modeling that included consideration of $PM_{2.5}$ as well as the gaseous pollutant NO_2. However, no results were provided in the paper for $PM_{2.5}$ in 1-P form so that a comparison on 1-P and 2-P results is not possible. Instead the authors reported that $PM_{2.5}$ was not significant when considered in combination with all the best predictors, based on a forward stepwise method of choosing explanatory variables.

[33] Korrick *et al.* (1998) is the one case where a multi-pollutant formulation appears to suggest that variance inflation is the root cause of $PM_{2.5}$'s lost significance, rather than the fact that the gaseous pollutant had greater statistical explanatory power.

Table 5. Summary of Sensitivity of Short-Term $PM_{2.5}$ Health Associations to Inclusion of a Gaseous Co-Pollutant (*)

Paper	City	Effect Estimated	Was any $PM_{2.5}$ coefficient significant?		Gaseous pollutant signif in 2-P?	Gaseous Pollutant
			1-P	2-P		
Delfino *et al.*, 1997	Montreal	ER visits	Yes	No	Yes	O_3
Sheppard, 2003	Seattle	Hosp adm	Yes	No (**)	Yes	CO
Lipfert *et al.*, 2000a	Philadelphia	Mortality	Yes	No	Yes	O_3
Korrick *et al.*, 1998	NH Mtns	Lung function indicators	Yes	No	No	O_3
Thurston *et al.*, 1994	Toronto	Hosp adm	Yes	No	Yes	O_3
Moolgavkar, 2003	Los Angeles	Hosp adm	Yes	No	Yes	CO, NO_2
		Mortality	Yes	No	Yes	CO
Delfino *et al.*, 1998	Montreal	ER visits	Yes	No	Yes	O_3
Peters *et al.*, 2000	E. Mass	Arrhythmia symptoms	Yes	No	Yes	NO_2
Fairley, 2003	Santa Clara Co, CA	Mortality	Yes	Yes	Yes for peak O_3	NO_2, O_3, CO
Gold *et al.*, 2003	Boston	HRV	Yes	Yes	Yes for O_3	O_3, NO_2, SO_2
Chock *et al.*, 2000	Pittsburgh	Mortality	No	No	No	Several

(*) Note: Zhang *et al.* (2000) also performed multi-pollutant modeling that included consideration of $PM_{2.5}$ as well as NO2. However, no results were provided in the paper for $PM_{2.5}$, either alone or in combination with other pollutants; the authors reported that $PM_{2.5}$ was not significant when considered in combination with all the best predictors, based on a forward stepwise method of choosing explanatory variables. It therefore cannot be included in Table 5.

(**) For Sheppard (2003), the reanalyzed GAM-based 1-P and 2-P results were both significant. However, the GAM code produces a biased standard error that overstates significance levels, and hence GLM-based results are viewed as more reliable and should be used when available. The GLM-based 1-P result is significant, while the GLM-based 2-P result in this paper is insignificant (albeit borderline), and the relative risk level is reduced. Additionally, all four of the seasonal coefficients for the 2-P GLM models are insignificant.

Table 6. Summary of Papers on Short-Term $PM_{2.5}$ Health Associations That Do Not Report Any $PM_{2.5}$ Results from 2-P Formulations that Included a Gaseous Co-Pollutant (*)

Paper	City	Effect Estimated	Is Paper Used in the Risk Analysis?	Is Paper Cited in Evidence Based Arguments of Proposed Rule?
Klemm & Mason, 2003; Schwartz, 2003a	Boston, MA	Mortality	X	X
	St. Louis, MO	Mortality	X	X
	Knoxville, TN	Mortality		X
	Madison, WI	Mortality		X
	Steubenville, OH	Mortality		X
	Topeka, KS	Mortality		X
Burnett & Goldberg, 2003	8 Canadian cities	Mortality		X
Ito, 2003	Detroit	Mortality	X	X
		Hosp adm	X	X
Mar *et al.*, 2003	Phoenix	Mortality	X	X
Smith *et al.*, 2000	Phoenix	Mortality		X
Clyde *et al.*, 2000	Phoenix	Mortality		
Schwartz and Neas, 2000 (**)	6 US cities	Resp symptoms	X	X
	State College, PA	Resp symptoms		X
	Uniontown, PA	Resp symptoms		X
Ostro *et al.*, 2003	Coachella, CA	Mortality		X
Tsai *et al.*, 2000	Elizabeth, NJ	Mortality		X
	Newark, NJ	Mortality		X
	Camden, NJ	Mortality		X
Peters *et al.*, 2001	Boston	Hosp adm		X
Goldberg & Burnett, 2003	Montreal	Mortality		X
Neas *et al.*, 1995	Uniontown, PA	Resp symptoms		X
Neas *et al.*, 1996	State College, PA	Resp symptoms		X
Tolbert *et al.*, 2000	Atlanta	ER visits		
Linn *et al.*, 1999	Los Angeles	Resp symptoms		
Liao *et al.*, 1999	Baltimore	HRV		
Ostro *et al.*, 1991	Denver	Resp symptoms		
Neas *et al.*, 1999	Philadelphia	Resp symptoms		X
Ostro *et al.*, 2001	So. Calif.	Resp symptoms		
Delfino *et al.*, 1996	San Diego	Resp symptoms		
Naeher *et al.*, 1999	SW Virginia	Resp symptoms		

(*) Note: Some of these studies did include 2-P models in an original paper that was affected by the GAM statistical errors. This summary does not account for any results that were not reanalyzed and reported in HEI (2003).

(**) Grant *et al.* (2002) list this paper as potentially having a GAM problem, but our review of this paper suggests this is not the case, and so it is listed here.

Flaws in the PR Case. Contrary to the evidence in Table 5, the PR states that recent short-term studies are "generally robust" to inclusion of gaseous co-pollutants.[34] I will now explain the flaws in the supporting evidence that the PR offers for this statement.

The first evidence the PR cites is two studies that did not include any measures of $PM_{2.5}$: Domenici *et al.* (2003) and Schwartz (2003b).

The PR then cites studies that did include $PM_{2.5}$, but the PR makes a more equivocal statement: "Similar results are seen in *some* single-city studies using $PM_{2.5}$ for *some* health outcomes in which the single-pollutant model association was statistically significant" (emphasis added).[35] EPA cites four papers to support this statement:

- Fairley (2003)
- Ito (2003) for hospital admissions for heart failure and pneumonia
- Sheppard (2003)
- Gold *et al.* (2003)

As Table 5 shows, Fairley (2003) and Gold (2003) do indeed find robustness to 2-P formulations. However, Sheppard (2003) finds that the association for $PM_{2.5}$ does become insignificant in a 2-P formulation with CO, when relying on the GLM results rather than the GAM results (as I noted in Table 5). Further, Ito (2003) does not report any 2-P results that included a gaseous pollutant simultaneously with $PM_{2.5}$, which is why it does not even appear in Table 5.[36]

As Table 5 shows, Fairley (2003) and Gold (2003) are just two of 11 studies in which both 1-P and 2-P results were reported. While this does qualify as "some" of the studies, when these two studies are placed in context of the total body of evidence, the case for robustness to inclusion of a gaseous pollutant is clearly incorrect.

The PR's discussion then diverts attention to the impact on the size of the $PM_{2.5}$ association. First, this ignores the more important implication, which is that the 2-P modeling suggests there may be no causal role of $PM_{2.5}$ at all in face of consideration of gaseous pollutants as well. Second, the supporting evidence EPA provides is miniscule: "The size of the effects estimates were little changed in other studies as well *in which the single-pollutant model associations were not statistically significant*" (emphasis added)[37] – and then they cite unspecified *insignificant* results in Ito (2003), which as already noted does not contain any

[34] PR, p. 2634.
[35] PR, p. 2634.
[36] The original paper that Ito (2003) reanalyzes did find a robust response to 2-P modeling, but reanalyzed 2-P results are not reported in Ito (2003). Ito (2003) at p. 153 does report 2-P results that included PM_{10} and $PM_{2.5}$ simultaneously, which is not the type of 2-P that is being discussed here. However, even so, these results reveal that the $PM_{2.5}$ associations for hospital admissions for heart failure and pneumonia are *insignificant* in these runs (and also insignificant for all other mortality and morbidity effect reported). The information provided in Figure 7 on Ito's p. 153 also reveals that the reanalysis with strict convergence for the GAM resulted in insignificance where it had once been significant for pneumonia admissions under the default GAM convergence criteria of the original study.
[37] PR, p. 2634.

2-P model results, and Chock *et al.* (2000), which happens to be the *single* study in Table 5 that did not find a significant 1-P effect! It is unsurprising that there would be little change between the 1-P and 2-P effects if the former is insignificant to start with.

The PR's statements regarding the robustness of $PM_{2.5}$ results to 2-P modeling goes on to note that "In yet other studies, however, for some combinations of pollutants in some areas, substantial reductions in the size of the effect estimates for $PM_{2.5}$ were observed."[38] For this they cite only Moolgavkar (2003), Thurston *et al.* (1994), and Delfino *et al.* (1998). Table 5 reveals that EPA could have cited five additional papers to support this statement: Delfino *et al.* (1997), Korrick *et al.* (1998), Lipfert *et al.* (2000a), Peters *et al.* (2000), and (arguably) Sheppard (2003).

EPA concludes by attempting to diminish the import of any findings based on 2-P models by stating that "collinearity between co-pollutants can make interpretation of such multi-pollutant model results difficult."[39] This is a baseless criticism, which may be why EPA provides no supporting citations. If collinearity were the cause of the sensitivity of the model results to 2-P formulations, then *both* the $PM_{2.5}$ and gaseous pollutant coefficients would face reduction in significance levels. However, Table 5 reveals that the gaseous pollutant remained significant in seven of the eight papers where the $PM_{2.5}$ effect was significant in the 1-P case but rendered insignificant in a 2-P case with a gaseous pollutant. Only the Korrick *et al.* (1998) paper would appear to reflect a problem of collinearity rather than a case of a gaseous co-pollutant having the more important explanatory power.

Thus, EPA has made an incorrect case that $PM_{2.5}$ associations are generally robust to inclusion of gaseous co-pollutants through the compounded effects of:

1. Referring to PM_{10}-only results;
2. Incorrectly citing two of four papers as evidence in favor of robustness;
3. Citing only three of eight papers that provide evidence of non-robustness; and
4. Arguing incorrectly that a statistical difficulty of collinearity complicates the findings of non-robustness, when the actual evidence reveals that to be a potential problem for only one of the eight papers finding non-robustness.

When all of the evidence is considered, the short-term studies reveal a substantial concern that a spurious association may be found for $PM_{2.5}$ if one relies on 1-P results. However, EPA's risk analysis and most of the figures in the PR showing results from individual papers rely solely on 1-P results, *even when 2-P results are available.*[40] Further, Table 6 reveals that the majority of $PM_{2.5}$ short-term epidemiological papers provide only 1-P results for $PM_{2.5}$ associations.

[38] PR, p. 2634.
[39] PR, p. 2634.
[40] Cases where the risk analysis relies on 1-P results but 2-P results are available are Moolgavkar (2003) for both mortality and morbidity in Los Angeles, Lipfert *et al.* (2000a) for mortality in Philadelphia, Fairley (2003) for mortality in San Jose, Chock *et al.* (2000) for mortality in Pittsburgh, and Sheppard (2003) for asthma in Seattle.

Long-Term Studies

It is also noteworthy that the long-term study on which EPA has based its quantitative risk analysis, that of the ACS cohort, has a well-established sensitivity to the inclusion of SO_2 in 2-P formulations with $PM_{2.5}$. Krewski *et al.* (2000) clearly demonstrated this was an issue with the 1-P results first reported by Pope *et al.* (1995):

> "We observed a stronger association between sulfur dioxide levels and mortality from all causes in the ACS Study than between either fine particles or sulfate and all-cause mortality....[T]he fact that sulfur dioxide was a stronger predictor of mortality than was sulfate does not appear to be due to the larger number of sulfur dioxide measurements. ...The sulfur dioxide effect on mortality risk was diminished for the best-educated subjects, a pattern we also observed with exposure to fine particles and sulfate. However, the sulfur dioxide effect, unlike the fine particle effect, was not the strongest for the least-educated subjects."[41]

The inclusion of SO_2 dramatically reduced the size of the estimated relative risk for $PM_{2.5}$, and rendered the $PM_{2.5}$ association statistically insignificant. "The inclusion of sulfur dioxide, which has a positive association with mortality (RR=1.30, 95% CI: 1.23-1.38) ... reduces the relative risk [for sulfate] from 1.16 to 1.04...[and causes] loss of formal statistical significance."[42] "The relative risk of all-cause mortality for fine particles, as with sulfate, was diminished after adjustment for ...sulfur dioxide (Table 37)."[43] Table 37 reveals that the $PM_{2.5}$ association falls from RR = 1.20 in the 1-P case to 1.03 in the 2-P case with SO_2. Additionally it goes from strong statistical significance in the 1-P case (CI: 1.11-1.29) to insignificance in the 2-P case (CI: 0.95-1.13).[44]

Despite this widely discussed finding, the extended analyses of Pope *et al.* (2002) do not report any 2-P results for $PM_{2.5}$ and SO_2, even though it does report that SO_2 has a significant association in its own 1-P formulation.[45] Importantly, EPA's risk analysis continues to rely on only 1-P results from ACS cohort studies, *including using the 1-P rather than 2-P result that is available in Krewski et al. (2000).* (The 2-P result for SO_2 is discussed only in a sensitivity analysis, combined with 2-P results for other gaseous pollutants that were not significant.) While there may be reasons, as EPA notes, to question the causality of the SO_2 association, that does not justify ignoring the observed sensitivity of the long-term mortality association to inclusion of this gaseous pollutant.

The other long-term study that EPA has used in its risk analysis, and uses in its evidence-based discussions is the "Six Cities Study" of Dockery *et al.* (1993), and reanalyzed by Krewski *et al.* (2000). This study, because of its very small number of cities, would not allow 2-P formulations to be tested. Technically speaking, this "was not practical because of the limited number of degrees of freedom (at most 6 *df*) for further analyses. The ACS Study,

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[41] Krewski *et al.* (2000), p. 224.
[42] Krewski *et al.* (2000), p. 179.
[43] Krewski *et al.* (2000), p. 181.
[44] Krewski *et al.* (2000), p. 184.
[45] Pope *et al.* (2002), p. 1140.

which involved 154 cities with a wide range of pollutant concentration profiles, was not seriously affected by this limitation."[46]

There are other long-term mortality studies that EPA does *not* rely on to build its case for tightening the $PM_{2.5}$ standard that also consider the impact of 1-P and 2-P formulations. Lipfert *et al.* (2000b) found no significant effect for $PM_{2.5}$ and mortality in any of a number of 1-P formulations.[47] They did find significant 1-P associations for O_3 and NO_2, which they subjected to 2-P formulations to check for robustness. These 2-P formulations (which used other PM indicators than $PM_{2.5}$) found that the gaseous pollutant associations were robust, and further, that O_3 appeared to be the dominant gaseous pollutant over NO_2.[48]

Flaws in the PR Case. I have already demonstrated significant flaws in the PR case that short-term $PM_{2.5}$ associations are robust to inclusion of gaseous co-pollutants. The PR briefly extends that argument to long-term studies with the single sentence: "Further, associations between long-term exposure to $PM_{2.5}$ and mortality were not generally sensitive to inclusion of co-pollutants, with the notable except of the inclusion of SO_2 in multipollutant models used in the reanalysis of the ACS study."[49]

This also is an incorrect summary of the evidence. The PR does recognize the non-robustness in the ACS study, but does not acknowledge that this is the *only* long-term mortality paper that EPA is relying on that reports any 2-P results. Its sensitivity to 2-P formulations is therefore a major concern. The PR also fails to recognize that gaseous co-pollutants had the robust association in the only other long-term mortality study that considered any 2-P formulations.

[46] Krewski *et al.* (2000), p. 216.
[47] In fact, almost all of the results find negative associations for $PM_{2.5}$ and mortality, most of which are statistically significant in the negative direction. This set of results alone should be given some weight in EPA's risk analysis and evidence-based approach, but is not.
[48] Lipfert *et al.* (2000b), pp. 60-61.
[49] PR, p. 2634.

Appendix A
Explanation of Basis for Robustness Ratings of $PM_{2.5}$ Studies

This appendix details the rationale used for summarizing the studies listed in Table 3 of my report in accordance with the classification system outlined as follows. I reviewed each study to determine the general significance level that it found for the $PM_{2.5}$ association specifically. I ranked them into one of three categories: "no overall significant association," "mixed significance" and "overall significant association." By "overall significant association," I mean that a majority of the regressions in the paper produced statistically significant associations. If a 2-P result is provided, it must also be statistically significant to be placed in this category, unless there is evidence of multicollinearity problems in the 2-P model.[1] A ranking of "no overall significant association" was assigned if the majority of the results in the paper are insignificant even if a statistically significant result exists in the paper. If there is only one 1-P and one 2-P result reported, and the 2-P is insignificant, I assigned it to this category, unless there is evidence of a multicollinearity problem in the 2-P result.

Table A.1 summarizes results in short-term mortality studies that included $PM_{2.5}$. Table A.2 summarizes results in short-term morbidity studies (including hospital admissions and emergency room visits.) Table A.3 summarizes results in studies of associations between symptoms and short-term $PM_{2.5}$ exposures.

In the ratings that appear in these tables the following numbering system is used:

1 = No overall statistically significant association in the study.

2 = Mixed statistical significance among models reported in the study.

3 = Statistically significant associations are the overall pattern in the study.

[1] Such evidence exists when both the PM and gaseous pollutant would become insignificant in a 2-P formulation even though both are significant in their respective 1-P formulations.

Table A.1 – Mortality Studies

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Ostro *et al.* (2003) – Coachella Valley, CA	1	15.8	33.4	Ostro *et al.* is a reanalysis of an earlier paper measuring the relationship between pollutants and mortality using a GAM model with stricter convergence and a separate GLM analysis. Authors state: "No association with cardiovascular mortality was found for PM2.5 or any of the gaseous pollutants" (p 201) and "[a]s in the original analysis, associations were detected for 0-day, 1-day, and 2-day lags for both PM10 and coarse particles, but not for PM2.5." (p 202). Accordingly, given the lack of significance, we have rated this measured association as a "1".
Schwartz (2003), Klemm and Mason (2003) – Knoxville, KY	1	20.8	43.5	Schwartz provides 5 mortality risk estimates for PM2.5 in Knoxville using a single-pollutant specification and finds two to be significant. Schwartz did not conduct a multi-pollutant analysis. Klemm and Mason have made risk estimates for the identical dataset using additional smoothing methods and find no significance in the risk estimates for Knoxville using a GLM model. Accordingly, given the lack of significance, we have rated this measured association as a "1".
Schwartz (2003), Klemm and Mason (2003) – Steubenville, OH	2	29.6	81.5	Schwartz provides 5 mortality risk estimates for PM2.5 in Steubenville using a single-pollutant specification and none were found to be significant. Schwartz did not conduct a multi-pollutant analysis. Klemm and Mason conducted risk estimates for the identical dataset, but using additional smoothing methods and similarly find no significance in the total risk estimates for Steubenville. However, using a GLM model Klemm and Mason find that PM2.5 has a significant association with COPD (3 instances) and with pneumonia in a single instance. Accordingly, given the significant association, specifically with COPD, we have rated this measured association as a "2".
Schwartz (2003), Klemm and Mason (2003) – Madison, WI	1	11.2	34.3	Schwartz provides 5 mortality risk estimates for PM2.5 in Madison using a single-pollutant specification and none were found to be significant. Schwartz did not conduct a multi-pollutant analysis. Klemm and Mason conducted risk estimates for the identical dataset, but using additional smoothing methods and similarly find no significance in the total risk estimates for Madison. Accordingly, given the lack of significance, we have rated this measured association as a "1".
Schwartz (2003), Klemm and Mason (2003) – Topeka, KS	1	12.2	32.0	Schwartz provides 5 mortality risk estimates for PM2.5 in Topeka using a single-pollutant specification and none were found to be significant. Schwartz did not conduct a multi-pollutant analysis. Klemm and Mason conducted risk estimates for the identical dataset, but using additional smoothing methods and similarly find no significance in the risk estimates for Topeka. Accordingly, given the lack of significance, we have rated this measured association as a "1".

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Ito (2003) – Detroit, MI	1	18.0	55.2	Ito contains six mortality risk estimates for PM2.5 in Detroit in a single-pollutant specification and all six are found to be insignificant. Although not reported in the paper, the authors estimated PM2.5 risks with many other models and a large portion of them apparently produced negative findings. Ito does not present a two-pollutant analysis. Given the lack of significance, we have rated this measured association as a "1".
Tsai *et al.* (2000) – Elizabeth, NJ	1	37.1	84.0	Authors explore the association between pollutants and total deaths and cardiovascular deaths in Elizabeth, NJ. Authors find no statistical association between FPM and the two death measures. Authors also investigated whether there was any association between deaths and FPM arising from various industries. They found no significant association between FPM and total deaths in all industries considered, though significance between FPM and two of the three industrial classifications for cardiorespiratory deaths. Given the over-all lack of significance, we have rated this measured association as a "1".
Moolgavkar (2003) – Los Angeles, CA	1	22.0	60.4	This paper contains 67 mortality risk estimates for PM2.5 in Los Angeles, plus a large number for other pollutants. Of these estimates for PM2.5, only six were found to be significant. A key point of the paper was to explore the relative roles of PM2.5 and other pollutants. Seven of the estimates were from two-pollutant models using CO and five of those seven were found to be insignificant for PM2.5. In contrast, CO was considerably more likely to be significant, where half of the 12 two-pollutant formulations that included CO were found to be significantly positive for CO. The author stated, "it is clear that CO was the best single index of air pollution associations with health endpoints, far better than ... PM2.5." (p 198) Given PM2.5's lack of significance, we have rated this measured association as a "1".
Chock, Winkler, and Chen (2000) – Pittsburgh, PA	1	20.5	56.3	This paper is primarily about PM10 risks as the authors downplay the usefulness of the PM2.5 results due to data limitations. The paper contains eight risk estimates for PM2.5 and all are found to be insignificant. The authors included other pollutants to explore their relative role and all other pollutants were found to also be insignificant. Given PM2.5's lack of significance as well as the lack of conclusions that can be drawn from these results, we have rated this measured association as a "1".

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Schwartz (2003), Klemm and Mason (2003) – Boston, MA	3	15.7	42.0	Schwartz provides 5 mortality risk estimates for PM2.5 in Boston using a single-pollutant specification and all five were found to be significant. Schwartz did not conduct a multi-pollutant analysis. Klemm and Mason conducted risk estimates for the identical dataset, but using additional smoothing methods and find less significance in the risk estimates for Boston than Schwartz. Klemm and Mason conduct additional estimates for total mortality risk and various sub categories using increasing degrees of statistical controls and find much lower and insignificant estimates in the most controlled formulation. However, given the overall high degree of statistical significance, we have rated this measured association as a "3".
Schwartz (2003), Klemm and Mason (2003) – St. Louis, MO	2	18.7	43.6	Schwartz provides 5 mortality risk estimates for PM2.5 in St. Louis using a single-pollutant specification and all five were found to be significant. Schwartz did not conduct a multi-pollutant analysis. Klemm and Mason conducted risk estimates for the identical dataset, but using additional smoothing methods and find less significance in the risk estimates for St. Louis than Schwartz. Klemm and Mason conducted additional estimates for total mortality risk and various sub categories using increasing degrees of statistical controls and find much lower and insignificant estimates in the most controlled formulation. Further, the vast majority of the estimates that do not use the GAM method were found to be insignificant. Accordingly, given the uncertainty over the appropriate level of statistical controls and the findings of the models not using uncorrected GAM, we have rated this measured association as a "2".
Lipfert *et al.* (2000) – Philadelphia, PA	2	17.3	44.2	This study provides 30 risk estimates for PM2.5 mortality risk. Of these 30 estimates, 14 effects were found to be significant. A key point of the paper was to explore the relative roles of PM2.5 and other pollutants. Seven of the estimates were from two-pollutant models using ozone and all were found to be insignificant for PM2.5 while the risk estimates for ozone were all significant, causing the authors to state: "The most immediate implications of this study are the apparent variability of the PM results and the robustness of peak O3 as predictors of daily mortality." Due to these mixed findings, we have rated this measured association as a "2".
Tsai *et al.* (2000) – Newark, NJ	2	42.1	94.2	Authors explore the association between pollutants and total deaths and cardiovascular deaths in Newark, NJ. Authors found a statistical association between FPM and the two death measures in Newark. Authors also investigated whether there was any association between deaths and FPM from various industries. They found a significant association between FPM and totals deaths in 3 of the 7 industries considered, and in one industry when considering cardiorespiratory death. Given the over-all level of significance combined with somewhat mixed findings at the industrial level, we have rated this measured association as a "2".

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Tsai *et al.* (2000) – Camden, NJ	2	39.9	84.4	Authors explore the association between pollutants and total deaths and cardiovascular deaths in Camden, NJ. Authors find a statistical association between FPM and the two death measures in Camden. Authors also investigated whether there was any association between deaths and FPM from various industries. They found a significant association between FPM and totals deaths in two of the seven industries considered, and in two industries when considering cardiorespiratory death. Given the over-all level of significance combined with somewhat mixed findings at the industrial level, we have rated this measured association as a "2".
Mar *et al.* (2003) – Phoenix, AZ	2	13.5	32.2	Mar *et al.* provide ten risk estimates for PM2.5 under a single-pollutant specification. Of these ten estimates, three were found to be significant. The authors considered the case of other pollutants (e.g. CO, NO2, SO2) and found similar results. The paper does not provide any risk estimates for two-pollutant formulations. Accordingly, due to these mixed findings, we have rated this measured association as a "2".
Fairley (2003) – Santa Clara County, CA	3	13.6	59.0	This paper provides 28 risk estimates for PM2.5 and of these estimates, 16 were found to be significant. Ten of these estimates are from single-pollutant formulations, two of which are significant. This paper also explores the relative roles of PM2.5 and other pollutants in two-pollutant models. The eighteen two-pollutant models started from a single-pollutant formulation that was significant for PM2.5. PM2.5 was found to be significant in 16 of the resulting two-pollutant specifications. The paper also explores seasonal patters in risk estimates and finds no pattern. On the strength of the findings of significance between PM2.5 and mortality, even in 2-P models, we have rated this measured association as a "3".
Goldberg and Burnett (2003) – Montreal, Canada	1	17.4	53.1	This paper of is a reanalysis of an earlier set of papers that used the GAM method, and the results in the current paper differ dramatically from the prior studies. The paper presents 16 estimates for an association between PM2.5 and non-accidental mortality using natural spline smoothing and all 16 are found to be insignificant. The paper also presents 36 estimates for an association between PM2.5 and various sub-groups of mortality, again using natural spline smoothing. Nearly all of these estimates, 35, are insignificant. The paper does not provide any multi-pollutant analyses. Given the complete lack of significance detailed in this paper, we have rated this measured association as a "1".

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Burnett and Goldberg (2003)	3	13.3	38.9	This paper is a reanalysis of an earlier paper by Burnett *et al.* (2000) though it is more limited in scope. The updated study describes 14 reanalyzed mortality risk estimates for PM2.5 pooled over all 8 cities. Two of these estimates used the GAM as in the original paper and were found to be significant. The remaining 12 included variations on the degree of smoothing and on the data included in the analysis, and in most of these cases PM2.5 was found to be significant. The paper does not provide any multi-pollutant analyses. On the strength of the findings of a significant association between PM2.5 and mortality, we have rated this measured association as a "3".

Table A.2 – Morbidity Studies: Hospital Admissions/Emergency Room Visits

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Delfino *et al.* (1997) – Montreal, Canada	1	12.1	31.2	Delfino *et al.* studied the association between emergency room visits for respiratory illnesses and air pollution in Montreal, Canada. Authors indicate that "[t]here were no significant associations between air pollutants and ER visits for patients in the age groups from 2 to 64 yr of age[.]" (p 571) PM2.5 with a one-day lag was shown to be significantly associated with ER visits for individuals over the age of 64 in a single-pollutant model, though this significance failed to be robust in a two-pollutant case with ozone included. In a separate analysis that controlled for weather and day-of-week trends, PM2.5 with a one-day lag was found to be significantly associated with an increase in the mean level of ER visit for individuals over the age of 64 in a single-pollutant case. Because PM2.5's significance in a single-pollutant model was found not to be robust to a multi-pollutant specification, we have rated this measured association as a "1".
Peters *et al.* (2001) – Boston, MA	3	12.1	28.2	Peters *et al.* examined the association between particulate air pollution and the onset of myocardial infarction (MI) for 772 patients in Boston, MA. Authors find that there is a mild significant association in a single-pollutant model between PM2.5 with a 1 and 2 hour lag and the probability of an onset of MI. No significance was found for four other lags considered or when using a 24 hour average PM2.5 level. PM2.5 was also found to be significant when the impact of 2 hour lag values and 24 hour average values were jointly considered. Because PM2.5 was found to be significantly associated with the onset of MI under multiple modeling assumptions, we have rated this measured association as a "3".
Sheppard (2003) – Seattle, WA	2	16.7	46.6	This study is a reexamination of results in a prior study focusing on the association between air pollution and asthma hospital admissions in Seattle, WA. Sheppard uses a GAM analysis with a more stringent default convergence criteria and also reports the results of a GLM analysis. In a single-pollutant analysis, there was a significant association between PM2.5 and hospital admissions under a one day lag. Under a two-pollutant analysis, PM2.5 appears to be only moderately significant and not significant under analyses of individual seasons. Because PM2.5 was found to be significant in a single-pollutant setting and moderately significant for some portion of the two-pollutant analysis, we have rated this measured association as a "2".

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Ito (2003) – Detroit, MI	3	18.0	55.2	This study is a reexamination of results in a prior study focusing on the association between air pollution and elderly hospital admissions in Detroit, MI. Ito uses a GAM analysis with a more stringent default convergence criteria and also reports the results of a GLM analysis. Ito restricts attention to single-pollutant analyses and reports findings at their most significant lag period. Ito finds PM2.5 to be significantly associated with hospital admissions for both pneumonia and heart failure. PM2.5 was not found to be significant for admissions for stroke, dysrhythmia, ischemic heart disease, and COPD. Because PM2.5 was found to be significantly associated with certain categories of hospital visits and no evidence was presented that contradicts those significant associations, we have rated this measured association as a "3".
Thurston *et al.* (1994) – Toronto, Canada	1	18.6[2]	51.0	Thurston *et al.* reports the results of an analysis of the air pollutants and daily hospital admissions in Toronto Canada. Authors find that there is a significant association between PM2.5 and total respiratory admissions in a single-pollutant analysis, but that PM2.5 loses its significance when included with O3 in a two-pollutant case, while ozone remains significant. Authors state "This points to the importance of considering as many pollutants as possible ... in order to diminish the chances of being misled as to which of the many ambient air pollutants is actually culpable for any noted air pollution-health effects associations." (p 282). PM2.5 was found not to be significant in either a single-pollutant or a two-pollutant case for total asthma admissions. Because PM2.5's significance in a single-pollutant model was found not to be robust to a multi-pollutant specification, we have rated this measured association as a "1".
Tolbert *et al.* (2000) – Atlanta, GA	1	19.4	41.5	The study presents the results of an GLM estimation of the impact of pollutants on adult asthma, COPD, dysrhythmia, and all-CVDs in Atlanta GA. Authors found an aggregated PM2.5 measure not to be significantly associated with any of the these four morbidity measures. Authors conducted a separate analysis of PM2.5 decomposed into five subclassifications – for asthma and COPD (ten scenarios in total) and none of these five PM2.5 definitions were found to be significant. For dysrhythmia and all-CVDs there were only three instances (out of ten total) where these PM2.5 definitions were significant. Given the limited degree of significance for PM2.5, we have rated this measured association as a "1".

[2] 18.6 is the average of the means for three years of data reported by the authors.

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Moolgavkar (2003) – Los Angeles, CA	1	22.0	60.4	The study is a reanalysis of results in three prior papers focusing on the association between air pollution and hospital admissions in Los Angeles. Moolgavkar uses a GAM analysis with a more stringent default convergence criteria and also reports the results of a GLM analysis. The morbidity endpoints considered were the impact on CVD and COPD. Under single-pollutant analyses, PM2.5 has a significant impact on daily CVD admissions with lags of zero and one day for both the restricted GAM and GLM models. However, this association for both lag days is eliminated in a two-pollutant case when CO is included in the analysis. The author finds similar results for COPD admissions. Thus, the significance of PM2.5 is not robust to the inclusion of additional significant explanatory variables and we have, therefore, rated this measured association as a "1".
Delfino *et al.* (1998) – Montreal, Canada	1	18.3	40.7	This study focuses on the association between respiratory illnesses and air pollutants among the elderly in Montreal Canada. The authors find that PM2.5 has no significant association with respiratory illness emergency room visits in any of the 3 cases considered (one single-pollutant case and two two-pollutant cases) causing the authors to remark: "Although there were adverse effects of estimated PM2.5 on ER visits for respiratory illnesses among the elderly, the association was unstable and completely confounded by both temperature and O3" (p. 74) Accordingly, we have rated this measured association as a "1".

Table A.3 – Morbidity Studies: Symptoms

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Peters *et al.* (2000) – Eastern Massachusetts	1	12.7	31.7	Peters *et al.* studied the association between air pollution and defibrillator discharges among 100 patients with such devices in eastern Massachusetts. For the six patients with ten or more discharge events, PM2.5 was significantly associated with discharges under a two day lag. No significance was found for same day, prior day, or three day lags or under a five day average pollutant level. For the 33 patients with at least one discharge event, PM2.5 was found not to be significant under any lag structure. Authors found NO2 to be significant under roughly half of the lag structures modeled. "Including both [PM2.5 and NO2] into one model reduced the effect estimate of PM2.5 effectively to 0, whereas the effect estimate of NO2 was unchanged." Accordingly, we have rated this measured association as a "1".
Korrick *et al.* (1998) – New Hampshire	2	15.0	41.2	This study measures the effects of ozone, PM2.5, and aerosol acidity on the pulmonary function of exercising adults in New Hampshire. The authors use mortality endpoints measuring expiratory volume and flow. In the single-pollutant case, the authors found PM2.5 to be significantly associated with three expiratory measures in the base case and in the adjusted case where age, sex, smoking status, etc are controlled for. However, all of these significant associations are eliminated in the two-pollutant case when ozone is included in the analysis. At the same time, separate multi-pollutant regressions of ozone, PM2.5, and acidity generate no significant association for ozone on pulmonary functions. We have rated this measured association as a "2".
Naeher *et al.* (1999) – Virginia	2	21.3	45.1	This study measures the effect of air pollution on daily changes in peak expiratory flow (PEF) in Virginia at two times during the day (morning and evening). In a single-pollutant analysis, the authors found PM2.5 to be significantly associated with morning PEF only as a same-day effect. PM2.5 was found to be marginally significant using a one day lag and using a 3 day average value. PM2.5 was found not to be significant for evening PEF. Under a multi-pollutant analysis for the morning PEF, PM2.5 was not found to be significant under any lag measure, prompting the authors to write: "When the main effects (H+ and PM2.5 or PM10) were analyzed as individual main effects in multivariate models related to morning PEF, none remained significant in the presence of any other." (p 120) Even though the significance of PM2.5 in the single-pollutant case was not found to be robust to a multi-pollutant specification, nor were the other pollutants found to be significant when considered in a multi-pollutant analysis. Thus, we have rated this measured association as a "2".

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Ostro *et al.* (1991) – Denver, CO	1	22.0	60.3	Ostro *et al.* measures the association between acidic aerosols and asthmatic response measured by general asthma status, cough, and shortness of breath. Authors found that PM2.5 was not significantly associated with any of these health measures. PM2.5 was found to be only moderately significant with respect to general asthma status when adjustments were made to control for missing values of PM2.5 in the data, prompting authors to state: "This study suggests that hydrogen ion is the pollutant of primary concern" and that "concentrations of particulate matter less than 2.5 microns were mildly associated with overall asthma ratings, but not with either cough or shortness of breath." (p 699) Accordingly, we have rated this measured association as a "1".
Neas *et al.* (1999) – Philadelphia, PA	1	22.2	44.9	Neas *et al.* measures the association between fine particulate matter and Peak Expiratory Flow Rate (PEFR) in children in Philadelphia, PA. Authors found under two standardized measures of PM2.5 (24-hour average and 5-day moving average) that PM2.5 was not significantly associated with reductions in PEFR in neither the morning or afternoon readings. Accordingly, we have rated this measured association as a "1".
Neas *et al.* (1996) Schwartz and Neas (2000) – State College, PA	2	23.5	69.0	Neas *et al.* measures the association between summertime haze episodes and PEFR and the incidence of various respiratory symptoms (cough, wheeze, or cold) in 108 children in State College, PA. Fine particulate matter was found to be significantly associated with the probability of experiencing a same-day cold but not on the probability of experience a same-day wheeze or cough. Under a one day lag, fine particulate matter was found to be significantly associated with the probability of a cough, but not of a wheeze or cold. Under both the same-day or one-day lag scenarios, fine particulate matter was not found to be significantly associated with a deviation in PEFR. Schwartz and Neas found PM2.1 to be nearly significantly associated with PEFR. Given these mixed findings, we have rated this measured association as a "2".
Neas *et al.* (1995) Schwartz and Neas (2000) – Uniontown, PA	3	24.5	60.0	Neas *et al.* measures the association between air pollutants and PEFR in 83 children in Uniontown, PA. Authors found that fine particulate matter was significantly associated with an increase in the probability of evening cough episodes. This continued to be the case under a separate scenario in which the amount of hours spent outdoors was controlled for. Fine particulate matter was nearly significantly associated with a decrease in the mean deviation in PEFR when time spent outdoors was controlled for but not significant otherwise. Schwartz and Neas found PM2.1 to be significantly associated with PEFR. Because PM2.5 was found to be significantly associated with the onset of a cough and there was no evidence provided that contradicted this result, we have rated this measured association as a "3".

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Ostro *et al.* (2001) – Los Angeles, CA	3	40.8	112.0	Ostro *et al.* measures the association between air pollution and asthma exacerbation measured as shortness of breath, cough, wheeze (symptoms), and the use of extra asthma medicine among African-American children in Los Angeles. For the asthma symptoms, authors conducted two analyses for the daily probability of each symptom and the probably of a new onset of a symptom episode, respectively (6 cases in total). PM2.5 was found to be significantly associated with asthma symptoms in all cases with the exception of the probability of a day-with-cough. The authors found no impact for any pollution on the use of extra asthma medicine stating: "With these models, none of the pollutants, pollens, or molds was associated with the use of extra medication among the full population. These null results were robust to model specification." (p 205) Accordingly, because PM2.5 was found to be significantly associated with the existence of asthma symptoms and there was no evidence presented that contradicted this result, we have rated this measured association as a "3".
Zhang *et al.* (2000) – Virginia	1	Not Provided	45.1	Zhang *et al.* measures the association between air pollution and respiratory symptoms, measured as a runny or stuffy nose, for a set of mothers in southwestern Virginia. Authors included PM2.5, PM10, Coarse PM, and other pollutants. The authors indicate that they ran a 2-stage estimation first "removing statistically insignificant terms (at the 0.05 level) from the model" (p 1210) prior to conducting the final model. In doing so, the final model included only Coarse PM and sulfate terms among the original set of potential explanatory pollutants, indicating that PM2.5 was found not to be significant under their methodology. Accordingly, we have rated this measured association as a "1".
Delfino *et al.* (1996) – San Diego, CA	1	24.8	51.1	Delfino *et al.* measured the association between ambient ozone and fine particle concentrations on daily asthma symptoms for sixteen children living in San Diego, CA. Daily asthma symptoms used in the analysis was measured as a composite score of five individual symptoms rated by participants in six levels of severity as well as the number of uses of an asthma inhaler each day. Authors report that "The low atmospheric levels of PM2.5, the SO4(2-) fraction of PM2.5, predicted aerosol H+, NHO3, and pollen were not associated with either asthma symptom scores or as-needed inhaler use." (p 638) Accordingly, we have rated this measured association as a "1".
Schwartz and Neas (2000) – Six Cities	3	18.0[3]	48.0	Schwartz and Neas measured the association between fine particles and respiratory symptoms in school children using the Harvard Six Cities Diary Study. Authors find that PM2.5 is significantly associated with lower respiratory symptoms under both single and double pollutant specifications. Given these findings, we have rated this measured association as a "3".

[3] Entry reflects median value. Mean not provided in study.

Study	Rating	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Linn *et al.* (1999) – Los Angeles, CA	1	24.8	59.1	Linn *et al.* measured the association between cardiovascular indicators and air pollutants on 30 individuals having severe COPD living in Los Angeles, CA. Measured cardiovascular indicators were arterial blood oxygen saturation, blood pressure, ECG readings and lung function. Authors find a highly significant association between peak flow and previous day's indoor PM2.5, though indicate that this association looses significance if outlying subjects are excluded. Authors find no significant relationship between blood oxygen saturation for any of the pollutants considered, though did find a significant association between overnight saturation and indoor PM2.5, though this association was found to be positive rather than negative as expected. Authors find no significant association between ECG readings and PM2.5 levels. Accordingly, given these findings we have rated this measured association as a "1".

Appendix B
Estimation of 98th Percentile Values for $PM_{2.5}$ Studies Cited in these Comments

The 98th percentile values for individual datasets play a central role in my comments on numerous $PM_{2.5}$ studies. The individual papers reporting $PM_{2.5}$-health associations rarely report this particular summary statistic for the $PM_{2.5}$ data that they used. These values were reported for fifteen of the studies cited in these comments in a January 28th, 2005 EPA memorandum (Ross and Langstaff (2005)). The PR commented on the 98th percentile levels of six more studies. After making a request to EPA, EPA released a supplemental memorandum on April 5, 2006 (Ross and Langstaff (2006)) that provided these data for six more studies. The EPA memos also report the annual averages for these 21 studies.

For remaining studies covered in my analysis, I estimated the 98th percentile ("98p") based on relationships derived from the relevant twenty-one studies for which the EPA memoranda specify statistical characteristics. I approached this in two ways, depending on the quality of air quality distributional data provided in the source papers.

If the source paper provided both a mean and standard deviation for their $PM_{2.5}$ data, then I estimated its 98th percentile by adding a multiple of the standard deviation to the reported mean. If the distributions of $PM_{2.5}$ were a normal distribution, the multiplier would be 1.96. However, $PM_{2.5}$ data are more skewed. I therefore used the EPA-provided data to determine a reasonable multiplier to use.[1] In particular, in six of the studies detailed in the EPA memoranda, the sample mean ("u") and standard deviation ("σ") are reported.[2] I computed the effective multiplier, Z, corresponding to those six 98th percentiles, where: $Z = (98p - u)/\sigma$. This returned values ranging from 2.37 to 2.96. Thus, for studies not covered by the EPA memorandum and for which authors provided summary mean and standard deviation statistics, I estimated their 98th percentile to be equal to: $98p = u + \text{Min } Z * \sigma$, where Min Z is the smallest Z value found among the data provided by EPA, that is, Min Z = 2.37. I relied on the smallest Z value to avoid overstating the 98th percentile. I have applied this estimation rule to 13 additional studies for which the authors reported both annual average (mean) and standard deviation statistics for their $PM_{2.5}$ data. These studies are noted in the accompanying table.

This left me with six studies in which authors did not report even a sample standard deviation statistic. All of these studies did report the mean $PM_{2.5}$, however. For these six, I relied on the average ratio of the 98th percentile statistic to the mean $PM_{2.5}$ concentration (ratio = (98p)/u) from the relevant studies detailed in the EPA Memorandum. The average value over all 21 studies reported by EPA is 2.62. Thus, for these remaining studies lacking standard deviation statistics, the 98th percentile is estimated to be: $98p = 2.62 * u$.

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[1] I first considered fitting a parametric lognormal to the datapoints provided, but discovered that a lognormal distribution produces much higher 98th percentiles than fit the 21 studies reported by EPA.

[2] The six studies are: Schwartz (2003) and Klemm and Mason (2003) for Madison, Topeka, and Boston, Lipfert *et al.* (2000), Mar *et al.* (2003), and Peters *et al.* (2001).

Tables B.1 through B.3 provide the precise data and calculations used to produce the 98th percentiles for the total of 37 datasets that I rely on in my comments.

Table B.1 – Mortality Studies

Study	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion of Percentile Calculation
Ostro *et al.* (2003) – Coachella Valley, CA	15.8	33.4	Value from Ross and Langstaff (2005). Attachment A reports this value as 33.8, but the source material from the author attached to that memo indicates the value is actually 33.4.
Schwartz (2003), Klemm and Mason (2003) – Knoxville, KY	20.8	43.5	Source paper reports standard deviation of 9.6. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 43.5 = 20.8+2.37(9.6)
Schwartz (2003), Klemm and Mason (2003) – Steubenville, OH	29.6	81.5	Source paper reports standard deviation of 21.9. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 81.5 = 29.6+2.37(21.9)
Schwartz (2003), Klemm and Mason (2003) – Madison, WI	11.2	34.3	Value from Attachment A in Ross and Langstaff (2005).
Schwartz (2003), Klemm and Mason (2003) – Topeka, KS	12.2	32.0	Value from Attachment A in Ross and Langstaff (2005).
Ito (2003), Lippmann *et al.* (2000) – Detroit, MI	18.0	55.2	Value from Attachment A in Ross and Langstaff (2005).
Tsai *et al.* (2000) – Elizabeth, NJ	37.1	84.0	Source paper reports standard deviation of 19.8. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 84.0 = 37.1+2.37(19.8)
Moolgavkar (2003) – Los Angeles, CA	22.0	60.4	The 98th percentile is estimated as the mean $PM_{2.5}$ level reported by authors multiplied by the average ratio (2.75) of the 98th percentile to particulate mean for studies reported in the EPA memoranda. Accordingly, 60.4=2.75(22.0).
Chock, Winkler, and Chen (2000) – Pittsburgh, PA	20.5	56.3	The 98th percentile is estimated as the mean $PM_{2.5}$ level reported by authors multiplied by the average ratio (2.75) of the 98th percentile to particulate mean for studies reported in the EPA memoranda. Accordingly, 56.3=2.75(20.5).
Schwartz (2003), Klemm and Mason (2003) – Boston, MA	15.7	42.0	Value from Attachment A in Ross and Langstaff (2005).

Study	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion of Percentile Calculation
Schwartz (2003), Klemm and Mason (2003) – St. Louis, MO	18.7	43.6	Source paper reports standard deviation of 10.5. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 43.6 = 18.7+2.37(10.5)
Lipfert *et al.* (2000) – Philadelphia, PA	17.3	44.2	Value from Attachment A in Ross and Langstaff (2005).
Tsai *et al.* (2000) – Newark, NJ	42.1	94.2	Source paper reports standard deviation of 22. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 94.2 = 42.1+2.37(22)
Tsai *et al.* (2000) – Camden, NJ	39.9	84.4	Source paper reports standard deviation of 18.8. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 84.4 = 39.9+2.37(18.8)
Mar *et al.* (2003) – Phoenix, AZ	13.5	32.2	Value from Attachment A in Ross and Langstaff (2005).
Fairley (2003) – Santa Clara County, CA	13.6	59.0	Value from Attachment A in Ross and Langstaff (2005).
Goldberg and Burnett (2003) – Montreal, Canada	17.4	53.1	Value from Attachment A in Ross and Langstaff (2005).
Burnett and Goldberg (2003)	13.3	38.9	Value from Attachment A in Ross and Langstaff (2005).

Table B.2 – Morbidity Studies: Hospital Admissions/Emergency Room Visits

Study	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Delfino *et al.* (1997)	12.1	31.2	Value from Attachment A in Ross and Langstaff (2005).
Peters *et al.* (2001)	12.1	28.2	Value from Ross and Langstaff (2006).
Sheppard (2003)	16.7	46.6	Value from Attachment A in Ross and Langstaff (2005).
Ito (2003)	18.0	55.2	Value from Attachment A in Ross and Langstaff (2005).
Thurston *et al.* (1994)	18.6[3]	51.0	Value from Attachment A in Ross and Langstaff (2005).
Tolbert *et al.* (2000)	19.4	41.5	Source paper reports standard deviation of 9.35. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 41.5 = 19.4+2.37(9.35)
Moolgavkar (2003)	22.0	60.4	The 98th percentile is estimated as the mean $PM_{2.5}$ level reported by authors multiplied by the average ratio (2.75) of the 98th percentile to particulate mean for studies reported in EPA memoranda. Accordingly, 60.4=2.75(22.0).
Delfino *et al.* (1998)	18.3	40.7	Source paper reports standard deviation of 9.5. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 40.7 = 18.3+2.37(9.5)

[3] Entry reflects median value. Mean not provided in study.

Table B.3 – Morbidity Studies: Symptoms

Study	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Peters *et al.* (2000)	12.7	31.7	Value from Ross and Langstaff (2006).
Korrick *et al.* (1998)	15.0	41.2	The 98th percentile is estimated as the mean $PM_{2.5}$ level reported by authors multiplied by the average ratio (2.75) of the 98th percentile to particulate mean for studies reported in the EPA memoranda. Accordingly, 41.2 = 2.75(15.0).
Naeher *et al.* (1999)	21.3	45.1	Source paper reports standard deviation of 10.1. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 45.1 = 21.2 + 2.37(10.1).
Ostro *et al.* (1991)	22.0	60.3	The 98th percentile is estimated as the mean $PM_{2.5}$ level reported by authors multiplied by the average ratio (2.75) of the 98th percentile to particulate mean for studies reported in the EPA memoranda. Accordingly, 60.3 = 2.75(22.0).
Neas *et al.* (1999)	22.2	44.9	Value from Ross and Langstaff (2006).
Neas *et al.* (1996) Schwartz and Neas (2000)	23.5	69.0	Value from Ross and Langstaff (2006).
Neas *et al.* (1995) Schwartz and Neas (2000)	24.5	60.0	Value from Ross and Langstaff (2006).
Schwartz and Neas (2000)	18	48.0	No summary statistic data is provided in this study. I have relied on summary statistics from Schwartz *et al.* 1994 the precursor study to Schwartz and Neas (2000) which are reported in Ross and Langstaff (2006).
Ostro *et al.* (2001)	40.8	112.0	The 98th percentile is estimated as the mean $PM_{2.5}$ level reported by authors multiplied by the average ratio (2.75) of the 98th percentile to particulate mean for studies reported in the EPA memoranda. Accordingly, 112.0 = 2.75(40.8).
Zhang *et al.* (2000)		45.1	No mean or standard deviation estimates provided. 98th percentile value assumed equal to Naeher *et al.* (1999) since both studies cover the same area (Southwest Virginia) and have the same reported minimum and maximums.
Delfino *et al.* (1996)	24.8	51.1	Source paper reports standard deviation of 11.1. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 51.1 = 24.8 + 2.37(11.1).

Study	Mean ($\mu g/m^3$)	98th Percentile ($\mu g/m^3$)	Discussion
Linn *et al.* (1999)	24.8	59.1	Source paper reports standard deviation of 14.5. 98th percentile value is computed assuming 98th percentile is 2.37 standard deviations above mean based on other data provided in EPA memoranda. Accordingly, 59.1 = 24.8 + 2.37(14.5).

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March 3, 2006

Stephen L. Johnson, Administrator
United States Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

RE: Proposed NAAQS for Particulate Matter

Dear Administrator Johnson:

The Children's Health Protection Advisory committee (CHPAC) appreciates this opportunity to provide comments to you on the proposed particulate matter standards. As the EPA's advisory panel on children's environmental health, we urge you to set the final National Ambient Air Quality Standards (NAAQS) for fine and coarse particulate matter at lower levels than proposed on December 20, 2005. While the proposal to lower the daily PM2.5 standard is a step in the right direction, the proposed standards do not provide adequate protection for infants and children. In addition, we urge you to extend coverage of the coarse particulate matter standard to rural areas, and to continue national monitoring of coarse particulate matter levels in both urban and rural areas. Finally, we urge you to reconsider exempting the agricultural and mining industries from regulation under the coarse particulate matter standard.

The mandate of the Clean Air Act is to set health-based standards for air pollutants at levels adequate to protect the public health, including the health of susceptible populations, with an adequate margin of safety. These principles have not only held up over time as the foundation of enormously effective public health interventions in air quality, they have also been upheld by the Supreme Court. The proposed standards do not provide an adequate margin of safety. In our letter of August, 8, 2005, we documented the many health effects of particulate matter on children, including exacerbation of asthma, reduced lung function, increased chronic respiratory symptoms, infant mortality, and adverse birth outcomes (Schwartz, 2004; AAP, 2004, U.S.EPA, 2005). These effects have been observed in a number of studies at exposure levels near and below the proposed standards.

We are especially concerned that it appears the health of children was neither adequately nor explicitly considered in determining the proposed standards, in particular with respect to the margin of safety considerations. Children breathe more air per body weight and per surface area of the lung than adults, are more active outdoors than adults, and there is likely higher deposition of particulate in the respiratory tract of children than adults (Phalen et al, 1985). Thus, children receive a higher dose than adults in the same setting. Furthermore, the respiratory tract is still developing postnatally, and is more vulnerable to insult than the adult lung. Finally, asthma prevalence and morbidity is higher in children than adults (Mannino et al., 1998), and asthmatics are especially susceptible to particulate matter pollution. These factors contribute to the adverse health effects observed in children at or below the level of the proposed standard. We strongly support the principle that the nation's children, who are especially susceptible to the harmful effects of air pollution, should be protected under the NAAQS.

The CHPAC has the following recommendations regarding the proposed Particulate Matter NAAQS.

1. Reduce the Proposed Annual Average Standard for PM2.5

Studies on health effects in children from chronic exposure to $PM_{2.5}$ provide evidence that children are not adequately protected by a standard of 15 $\mu g/m^3$. A study of children in Los Angeles demonstrated that long-term exposure to $PM_{2.5}$ (mean across communities about 15 $\mu g/m^3$) was significantly associated with clinically reduced lung function at age 18 years (Gauderman et al., 2004), which is likely to be an irreversible effect. A number of studies of traffic-related pollution have shown associations between fine particles and adverse respiratory outcomes, including asthma in children who live near major roadways (van Vliet et al., 1997; Brunekreef et al., 1997; Kim et al., 2004), with mean annual average fine particle concentrations near and below 15 $\mu g/m^3$. The Harvard 24-cities study (Raizenne et al., 1996) showed effects on children's lung function at a mean of 14.5 $\mu g/m^3$.

The EPA based its annual standard for $PM_{2.5}$ on mortality studies in adults because of the robust nature of the data. In evaluating studies of the health effects of chronic exposure to particulate matter, EPA staff use the mean of the measured chronic exposure levels as the approximate effects level. The mean exposure level across a number of studies demonstrating health effects in children, including those cited above, is at or below the level of the proposed annual $PM_{2.5}$ standard of 15 $\mu g/m^3$. Thus, the proposed annual standard does not provide the required adequate margin of safety to protect infants and children.

2. Reduce the Proposed Short-term Standard for PM2.5 .

The proposed 24-hour average (daily) standard for $PM_{2.5}$ of 35 $\mu g/m^3$ (98^{th} percentile form), which is based on studies in adults, will leave a significant number of children unprotected from short-term effects on respiratory health. Several investigations demonstrate adverse respiratory health effects in children at daily levels (upper percentiles) near the proposed short-term standard, including respiratory hospital admissions, decreased lung function, asthma exacerbations, and respiratory symptoms

(Delfino et al, 1997; Tiittanen et al, 1997; Norris et al, 1999; Schwartz and Neas, 2000; Delfino et al., 2002; Delfino et al., 2004; Lewis et al, 2005; Barnett et al, 2005). Additional studies have found significant elevations in adverse birth outcomes including prematurity (Ritz et al., 2000; Sagiv et al, 2005; Wilhelm and Ritz, 2005), low birth weight or small–for-gestational age (Ha et al., 2001; Wilhelm and Ritz, 2003, 2005; Parker et al., 2005) and heart defects (Gilboa et al., 2005), as well as elevated risk of infant mortality (Loomis et al., 1999; Bobak and Leon, 1999; Lipfert et al., 2000; Ha et al., 2003; Woodruff et al., 1997, 2006) in association with measures of daily PM_{10} or $PM_{2.5}$. In some studies, the upper percentiles of the distribution of daily PM were close to or below the proposed daily $PM_{2.5}$ standard of 35 $\mu g/m^3$ (98^{th} percentile form). We urge the Administrator to take into consideration the serious health effects reported in these studies and revise the daily $PM_{2.5}$ standard downward to protect children's health.

3. Reduce the Short-term Coarse PM (PM10-2.5) Standard

The Notice of Proposed Rulemaking (NPRM) proposes a coarse PM ($PM_{10-2.5}$) short-term 24-hr average standard of 70 $\mu g/m^3$ (98th percentile form). This level does not provide an adequate margin of safety to protect children. In addition, there is no justification to exclude children who live in areas with populations below 100,000 from protection under the coarse particle standard, or to cease monitoring for coarse particles in these areas.

Level of the standard

While the studies cited in the NPRM as the basis for the coarse PM standard looked at morbidity and mortality in adults, the coarse fraction of PM ($PM_{10-2.5}$) has been associated with several respiratory outcomes in children, including significant associations with asthma hospitalizations (Lin et al., 2002), respiratory hospitalizations (Lin et al., 2005; Yang et al. 2004; Burnett et al., 2001), cough (Tiittanen et al., 1999; Schwartz and Neas, 2000; Mar et al., 2004), persistent cough, persistent phlegm and bronchitis (Zhang et al., 2002). Concentrations of daily mean $PM_{10-2.5}$ in these studies range from 6 to 59 $\mu g/m^3$. In some, the upper percentiles of daily $PM_{10-2.5}$ are well below the proposed standard of 70$\mu g/m^3$. A recent review of over 30 studies (many published prior to 2003) that evaluated both fine and coarse PM notes that, in many studies, coarse PM is related to respiratory morbidity, including hospital admissions for children, more strongly or at least as strongly as fine PM (Brunekreef and Forsberg, 2005). The NPRM notes that deposition of coarse particles is higher in the tracheobronchial region of the lung, which is a critical target in asthmatics. The proposed standard of 70 $\mu g/m^3$ does not adequately take into account the coarse particle studies that have observed serious health effects in children.

Rural versus urban

The NPRM states EPA could not confirm or refute effects of crustal coarse PM ($PM_{10-2.5}$). The EPA's response to this uncertainty has been to exclude rural areas (areas, including small cities, with populations less than 100,000), presumably under the assumption that rural PM is dominated by crustal sources, from coverage under the standard. Exclusion of cities and regions from coverage based on number of residents is an unprecedented departure from previous practice under the NAAQS program and runs

counter to the purpose of a national standard. Limiting the $PM_{10-2.5}$ standard to urban areas is not scientifically supportable and is not in the interests of the health of children living in small cities and rural areas. It should be noted that wind-blown dusts can contain toxic elements. For example, arsenic, cadmium, and nickel are in high concentration in the soil of the Owens Valley in California, an area with high coarse PM levels. Crystalline silica, a common constituent of rural dust, is a human carcinogen and can cause silicosis at relatively low levels in occupational settings. Chronic silicosis has been described after environmental exposures to silica in regions where soil silica content is high and dust storms are common (ATS, 1997). Thus, there is insufficient data to reasonably conclude that there is no need to regulate rural $PM_{10-2.5}$.

Exemption of agriculture and mining industries

The EPA has categorically exempted agricultural and mining sources of coarse PM from the proposed standard. Since under the Clean Air Act, implementation is not to be considered in setting standards, this exclusion could only be justified if there is no basis from a public health standpoint to control particulate matter emissions from these sources. There is insufficient and unpersuasive scientific evidence to support this action, nor was it supported by the Clean Air Science Advisory Committee (CASAC) review.

Monitoring of coarse particles

The EPA concluded that there is uncertainty with respect to the risk posed by rural coarse PM. To the extent that such uncertainty exists, the EPA proposal to decrease monitoring for coarse particles in rural areas prevents future scientific studies that would be able to resolve this uncertainty. We urge the Administrator to require ongoing monitoring of the coarse PM fraction in rural areas.

Conclusions and Recommendations

The CASAC and the EPA staff paper have both made recommendations that support more health protective standards than those proposed in the NPRM. The CHPAC urges you to revise the proposed $PM_{2.5}$ daily and annual standards and the $PM_{10-2.5}$ daily standard downward to the lower end of the ranges recommended in the EPA staff paper and by CASAC, to extend coverage of the coarse PM standard to rural children, and to continue monitoring coarse PM in both rural and urban areas. We thank you in advance for considering these comments, and would be happy to discuss these comments with you or your staff.

Sincerely,

Melanie A. Marty, Ph.D., Chair
Children's Health Protection Advisory Committee

Cc: William Wehrum, Designated Assistant Administrator, Office of Air and Radiation
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Lydia Wegman, Office of Air Quality Planning and Standards
Dr. William Sanders, Interim Director, Office of Children's Health Protection and Environmental Education
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Docket

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April 17, 2006

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Re: Proposed Coarse PM NAAQS
(Federal Register 71:2620-2708)

Dear Administrator Johnson and Acting Administrator Wehrum:

I have prepared the following comments at the request of the National Cattlemen's Beef Association and the National Mining Association in order to share my concerns about the scientific basis for the proposed NAAQS for coarse particulate matter ($PM_{10-2.5}$) that was recently published in the *Federal Register* (*Fed Reg* 71:2620, 01/17/06).

To introduce myself, I have attached a short summary of my experience and qualifications in medicine, epidemiology, toxicology and occupational health science. I am Associate Clinical Professor of Medicine and Epidemiology & Public Health at Yale University. I teach required graduate courses in both Toxicology and Risk Assessment. I also served for 10 years as a founding member of EPA's National Advisory Committee on Acute Exposure Guideline Levels for Hazardous Substances (NAC/AEGL).

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This is the third set of comments that I have submitted on this issue. Twice during the past year, in May and August, I submitted comments to the Clean Air Scientific Advisory Committee. Then as now, my principal concern is the general lack of scientific support for a coarse particulate matter standard and the failure of EPA to appropriately address that deficiency.

Those limitations were acknowledged in various versions of the Staff Paper (e.g., EPA-452/R-05-005, June 2005):

> "a growing, but still limited, body of evidence on health effects associated with thoracic coarse particles from studies that use $PM_{10-2.5}$ as a measure of thoracic coarse particles." (Final Staff Paper: 5-47)

That concern is restated word-for-word in the Proposed Coarse PM NAAQS:

> "In developing this rationale, EPA has taken into account the information available from a growing, but still limited body of evidence on health effects associated with thoracic coarse particles from studies that use $PM_{10-2.5}$ as a measure of thoracic coarse particles." (*Fed Reg* 71:2653)

The Preamble also acknowledges that similar concerns were expressed by the CASAC, which noted:

> "... significant uncertainties resulting from the limited number of studies to date in which $PM_{10-2.5}$ has been measured and the potentially large exposure measurement errors in such studies". (*Fed Reg* 71:2671)

As discussed below, the actual evidence available to support the Proposed Coarse PM NAAQS is substantially more limited than is acknowledged by EPA. The relative insufficiency of evidence linking coarse particulates to human health effects is repeatedly acknowledged in the Proposed Coarse PM NAAQS. For example, consider Section III.A, which describes the Evidence of Health Effects Related to Thoracic Coarse Particle Exposure. In that very important, but relatively short section (it comprises only 10 pages of the Preamble), EPA reiterates 24 times that the evidence linking coarse particulate to health effects is either "limited" or "very limited". By contrast, EPA does not once describe any of the evidence as "sufficient" or "adequate".

Despite that apparent recognition of these limitations, however, EPA presents the actual data from cited studies in a manner that overstates their informational value. More worrisome is the possibility that the Preamble has been constructed in a manner intended to obscure the deficiencies and to minimize objections that might be raised about the lack of scientific justification for the Proposed Coarse PM NAAQS.

1). For example, the Preamble discusses the most important limitations of evidence in a brief section (*Fed Reg* 71:2671-2), distanced from the primary presentation of the cited studies and their data, and relegated mainly to discussion of an "alternative

interpretation" that is strikingly devoid of specific details. Thus, notwithstanding repeated statements about data limitations, many readers will fail to appreciate the actual magnitude of the deficiency of scientific evidence.

a) My concerns are illustrated by the following example. The centerpiece of the presentation of the Evidence of Health Effects is Figure 2 (page 2656), which summarizes the "Effect estimates for associations between short term exposure to $PM_{10-2.5}$ and mortality or morbidity health outcomes ..." The legend to Figure 2 states:

> "for consistency across studies, effect estimates are from single-pollutant, general linear models".

The decision that only data from single-pollutant models would be presented in this centerpiece graphic is at least curious because the compiled literature provides good evidence that single-pollutant models overestimate the effects of coarse particulate. The possibility that such an approach should not be taken, however, is only discussed in the context of the "alternative interpretation":

> "... an alternative interpretation of the available health evidence presented in the Criteria Document and the Staff Paper ... suggests that that the results from one-pollutant $PM_{10-2.5}$ models are confounded by fine particles and gaseous co-pollutants... Taken as a whole, evidence from $PM_{10-2.5}$ epidemiologic studies could be interpreted to suggest that one-pollutant $PM_{10-2.5}$ models suffer from bias due to omitting co-pollutants in the statistical model..." (*Fed Reg* 71: 2671-2)

But even that statement does not reasonably represent the scientific evidence. It implies that this is solely a matter of "interpretation", as though reflective of a philosophical debate. In fact, numerous studies cited in the Proposed Coarse PM NAAQS provide evidence that single-pollutant models overstate the apparent risks of coarse particulate. I am aware of none that proposes the contrary.

For example, consider the Toronto study by Burnett et al. (1), which is cited eight times in the Preamble. That study found that positive associations noted in a single-pollutant model "disappeared after adjustment for O_3, NO_2, and SO_2" (1). But Figure 2 presents only the results of a single-pollutant model from that study, thus wrongly indicating a statistically significant effect of coarse particulate [1]. Or, consider that use of two-

[1] More surprising is footnote #52 (p. 2657), which disclaims the analytical findings of the multiple-pollutant model in Burnett et al. The footnote states that their results "show relatively consistent effects estimates ... except for the models including NO_2 and all four gaseous pollutants". This footnote implies a preference for relying on an incomplete analysis of a complex dataset. Of greater concern is that EPA has apparently discounted the authors' finding that the association "could be completely explained by NO_2, a risk factor not as widely considered in North American locales as the other criteria pollutants" (1). Similar findings and cautionary advice are found in Thurston et al. (12); significant associations with coarse particulates "were merely a statistical by-product of inter-pollutant confounding ... This points out the importance of considering as many pollutants as possible in such analyses, in order to diminish the chances of being misled..."

pollutant models including both $PM_{2.5}$ and $PM_{10-2.5}$ reduced or eliminated the estimated effects of coarse particulate in the Six Cities study (2) and in studies of Detroit (3,4), Los Angeles (5) and eight Canadian cities (6) [2].

I am not alone in pointing to the need to consider multiple-pollutant models in order to correctly understand the effects of coarse particulate. That approach was stressed by the Research Committee of the Health Effects Institute in comments on the Detroit study conducted by Lippmann et al. (3):

> "In order to determine the relative effects of several risk factors on a health outcome, ideally all variables under considerations would be included in a single model."

In the apparent pursuit of "consistency", EPA has selectively presented the least rigorous of the available evidence, thereby minimizing its informational value. Even for those studies which provided results from dual- and/or multiple-pollutant models, EPA has emphasized single-pollutant analyses while discounting the data from more rigorous multi-pollutant analyses. In so doing, EPA has systematically overstated the apparent effects of coarse particulate.

Had EPA correctly acknowledged that the results of single-pollutant models generally overestimate the effects of coarse particulate and that most of the cited studies provided only results of such models, then the even more limited nature of evidence here would have been readily appreciated.

2). Following is another example of the failure to describe and respond to limitations of the evidence presented as justification of the Proposed Coarse PM NAAQS.

In the Final PM Staff Paper (EPA-452/R-05-005, June 2005), EPA Staff described a criterion for deciding whether studies effects data were sufficiently precise to be used in quantitative estimates of exposure-response relationships. In so doing, Staff correctly recognized that some studies are better than others (because of their size or presumably for other reasons) and that studies of lesser quality should not be relied upon as one might rely on studies of higher quality. In particular, the Staff Paper described a "rough indicator of ... precision" that was used for this purpose:

> "The natural logarithm of the mortality-days (a product of each city's daily mortality rate and the number of days for which PM data were available) can be used as a rough indicator of the degree of precision of effect estimates ... staff

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The literature cited by EPA is dominated by studies with analytical models that failed to consider other pollutants and risk factors. Thus the conclusions of Burnett et al. and Thurston et al. give added reasons to view the EPA evidence with caution. I do not agree with the Preamble statement that "effect estimates for associations between PM, including $PM_{10-2.5}$, and health endpoints are generally robust to confounding by gaseous co-pollutants" (*Fed Reg* 71:2660}.

[2] These studies are all cited in the Preamble. Results of the Los Angeles and the eight Canadian cities studies were excluded from Figure 2 because they used GAM, rather than GLM analytical models.

> chose to consider only those urban areas in which studies with relatively greater precision were conducted, specifically including studies that have a natural log of mortality-days greater than or equal to 9.0 (i.e., approximately 8,000 deaths) for total non-accidental mortality." (SP, p. 4-6)

That approach (both specifically and generally) has been deleted from the Proposed Coarse PM NAAQS. As a result, EPA has deleted its Staff's criterion for objectively distinguishing between individual studies. It is interesting to note that if EPA had accepted this criterion, then it would have had to acknowledge that results from the Coachella Valley studies (7-9) and the Six Cities study results from Steubenville (10,11) had been judged to be of "lesser quality".

But the Preamble relies on those two studies repeatedly: The Coachella Valley studies are cited 19 times, while the Steubenville data are cited eight times. At no point does the Preamble indicate that EPA Staff had objectively determined that both data were too imprecise to be used for quantitative assessments and thus their conclusion should be viewed with caution. I am concerned that the failure to indicate those Staff determinations serves mainly to conceal the limitations of those studies [3].

3). A third example of the failure to describe and respond to limitations of the evidence relates to the adequacy of the exposure assessments that underlie each of the individual studies. Concerns about the precision and accuracy exposure assessment can not be separated from concerns about the precision and accuracy of the studies themselves.

a) One aspect of my concern involves the spatial location(s) of monitors used to describe the exposures of study populations. It is generally accepted that coarse PM (e.g., $PM_{10-2.5}$) deposits more rapidly and more locally than does fine particulate. Likewise, it is generally accepted that local sources are of greater importance in determining concentrations of coarse particulate (6). Accordingly, it can be expected that measurements from centrally located monitors will less accurately represent regional exposures to coarse particulate than fine particulate (i.e., $PM_{2.5}$). For that reason, measurements of coarse PM obtained at relatively distant monitoring stations should be viewed with caution, and so should studies that rely on coarse PM measurements obtained relatively far from target populations. When such distant measures are used as the basis for epidemiological studies, efforts should be made to demonstrate that the distant measures do accurately reflect the exposures of target populations.

For example, in an analysis and comments submitted separately, Gale Hoffnagle describes marked spatial variation of fugitive coarse PM emitted by ground level sources such as those characteristic of agricultural and mining activities. His analysis indicates that even when levels at such sources reach several hundred mg/m^3, corresponding levels at a distance of 1000 meters are *de minimis* (i.e., they approach zero mg/m^3). Thus PM

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[3] It is also notable, and perhaps related, that despite a statement in footnote 50 (*Fed Reg* 71:2655) that two subsequent reanalyses of the Steubenville data found essentially no significant associations, the Preamble persists in referring to the original Steubenville data as showing "a statistically significant mortality association".

monitors located at a distance of 1000 meters or more reflect little or no contribution from such sources.

However, a number of the studies cited in the Preamble depended on coarse PM measurements from distant monitors and were apparently not accurate predictors of target population exposures. In the Detroit study by Lippmann et al. (3,4), particulate matter data were obtained from ambient monitors in Windsor, Ontario, several miles from central Detroit. The Staff Paper and the Proposed Coarse PM NAAQS document that levels measured in Windsor were not representative of those in Detroit:

> "In recent years, based on available Windsor and Detroit data from 1999 to 2003, the Windsor monitors used in this study typically have recorded $PM_{10-2.5}$ levels that are generally less than half the levels recorded at urban-center Detroit monitors..." (Staff Paper: 5-68)

Accordingly, on the basis of that exposure concern, the Detroit study must be regarded as providing only limited informative value.

In the Coachella Valley studies (8,9), particulate measures were obtained in Indio, approximately 25 miles from older population centered in the Palm Springs area at the western end of the Valley [4].

b) A second exposure assessment concern is the manner in which coarse particulate levels are determined. The Preamble notes that $PM_{10-2.5}$ measurements are prone to greater exposure errors than are measurements of $PM_{2.5}$ (*Fed Reg* 71:2660). In addition, $PM_{10-2.5}$ levels calculated by the difference method (i.e., subtracting $PM_{2.5}$ from PM_{10}) can be expected to have larger errors than $PM_{10-2.5}$ levels directly measured using dichotomous samplers; the difference method is impacted by two measurement errors, while the direct measurement method has only one. And when the difference method is performed using data from monitors that are not physically co-located, additional exposure assessment errors result because of non-homogeneous spatial distributions of particulate matter.

Finally, estimation of coarse particulate exposures derived from only PM_{10} measurement in areas where measured PM levels are "dominated" by coarse particulate are by far the most uncertain and least accurate. Because of such uncertainty, the findings of epidemiological studies that rely on those exposure assessments should be viewed as the least informative, analogous to ecological studies that are suitable for generating, but not testing hypotheses.

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[4] The population of the Indio area, which is on the Northern rim of the Valley, differs from that of many of the other Valley communities. For example, according to the 2000 Census, 15.2% of the population was in the 45-64 year age group and 9.1% were over 65 years. By contrast, the corresponding proportions were 26.4% and 26.2% for Palm Springs, 30% and 43% for Ranch Mirage and 26.3% and 27.6% for Palm Desert. Those cities, with significantly older populations more prone to cardiorespiratory diseases, are located approximately 10-25 miles away toward the Western end of the Valley.

Consider the effect of categorizing the studies cited in the Preamble on the basis of their exposure assessments.

a) The highest quality $PM_{10-2.5}$ exposure assessments are those in studies that employed dichotomous samplers.

Dichotomous particulate samplers were used in two Toronto studies (1,12) that considered hospital admissions and two reports from the Harvard Six Cities study, one considering mortality effects (10,11) and other peak flow and asthma in children (2). All four of those studies found no significant effects associated with exposure to $PM_{10-2.5}$.

b) Second tier studies calculated $PM_{10-2.5}$ by the difference method, subtracting $PM_{2.5}$ from levels of PM_{10}. Among the co-location studies cited in the Proposed Coarse PM NAAQS, most suffered important data limitations or deficiencies.

No association of respiratory symptoms and childhood asthma were found for coarse particulate calculated by the difference method in Uniontown and State College (2). There were only marginal associations ($0.05<p<0.10$) between coarse particulate calculated by the difference method and mortality in the Phoenix study (13,14). The Detroit study found small positive associations for coarse particulate but as discussed above, particulate data were obtained miles from the study population and were significantly inaccurate (3,4). The HEI Health Review Committee concluded that data from the Detroit study were inconclusive:

> "...the data do not clearly support a greater effect of one pollutant over another, nor do they establish which pollutants are most likely to cause adverse health effects..." (3) (HEI Synopsis)

The Coachella Valley study (8,9) reported positive associations with mortality and the Seattle study (15,16) reported positive associations with hospital admissions for asthma in non-elderly patients. But both studies suffered from large data gaps that were filled by imputation and arbitrary calculations; in both studies, exposure data were missing for 75% or more of the $PM_{2.5}$ values and, therefore, they were also missing for coarse particulate exposure measures [5].

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[5] In the Coachella Valley study, PM_{10} data were available for a 10-year period, but $PM_{2.5}$ data for only 2.5 years. The missing $PM_{2.5}$ and $PM_{10-2.5}$ were imputed using a predictive function that estimated $PM_{10-2.5}$ as a cubic function of PM_{10}. The predictive function was such a poor fit for $PM_{2.5}$ data that the authors concluded that "predictive models could not be successfully estimated" (9). Accordingly, the calculated values, which represented 75% of the $PM_{10-2.5}$ data, can not be viewed as reliable.

The extent of missing data in the Seattle study is no less extreme. The authors observed: "Numerous missing PM measurements potentially limit our analysis" (15). For the three monitoring stations considered, no $PM_{2.5}$ data were available for 72-100% of days. The authors "imputed" the missing data. The imputation methods were not described, but the authors indicate that six different imputation methods were used and the results of those six methods were averaged. In addition, the "exposure" data were then "weighted" to favor residential areas, but no justification for that arbitrary weighting scheme was

c) The lowest tier studies measured only PM_{10} in areas thought to be dominated by coarse particulate and thereby inferred associations with coarse particulate. The lowest tier studies included "positive" studies in Anchorage (17,18,18), Reno (19), Tucson (20), and the Coachella Valley (7). Each also suffered from additional methodological concerns.

In Anchorage (17,18), the health effects were measured in terms of outpatient visits, not episodes of illness, and included events likely to be primarily infectious (e.g., "sore throat, ear aches"). Repeated visits by the same individual (e.g., emergency visits and follow-up office visits) would result in temporal dependence among outcomes that would effectively underestimate variance and overestimate the significance of associations, perhaps leading to inappropriate rejection of the null hypothesis of no effect of particulate exposures. In addition, outcome measures were not associated with the highest levels of exposure, only with lesser exposure levels.

The Reno study (19) provided no evidence that PM_{10} was dominated by coarse particulate. However, two facts suggest that PM_{10} was dominated by fine particulate, not coarse particulate. First, PM_{10} levels were inversely related to wind speeds, suggesting that those levels reflected not wind-blown crustal particulate, but decreased dispersion of suspended fine particulate. Also, the authors noted that:

> "Higher peaks occurred during the winter season. This may be as a result of increased residential combustion due to cold weather in the study area." (19)

If the authors are correct, then those peaks would have represented fine, not coarse particulate. Accordingly, the relevance of the Reno study to coarse particulate exposures is uncertain at best.

The Tucson study, which evaluated cardiovascular hospitalizations, used data from a monitoring station that was "located in a neighborhood site likely to be representative of population exposure", rather than at a site that would have been "subject to windblown dust" (20). Therefore, it is likely that the Tucson PM_{10} exposure data derived from samples that were actually dominated by fine particulate. Unfortunately, there were no $PM_{2.5}$ data are available to validate the underlying assumptions of this exposure assessment.

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provided. Thus, the "exposure data" in this study were mainly synthetic, rather than empirical, and had been transformed in ways that can not be understood and have not been justified. It is difficult to regard this as a valid observational study.

> The Coachella study (7) utilized PM_{10} data from monitors located in Indio, 25 miles from the major population center around Palm Springs, where on average PM_{10} levels were 21% greater than in Indio. Given the higher population and vehicular density in Palm Springs, it seems likely that the higher levels reported during the study period in Palm Springs reflected mainly fine combustion particulate, rather than windblown crust.

By means of such a categorization scheme, it can be seen that most of the evidence in support of the Proposed Coarse PM NAAQS is derived from studies with the lowest quality of exposure assessments, while those with highest quality exposure assessments lend no support.

If EPA had used such a 'quality of exposure assessment' approach to prioritize the evidence available [6], it would have been apparent that support for the Proposed Coarse PM NAAQS is mainly found in the least robust studies. Thus, such an approach would have further emphasized the limitations of supporting evidence.

4). In summary, EPA has systematically presented the results of cited studies in a manner that overstates the evidence linking coarse particulate and health effects.

> Data from inferior single-pollutant models have been presented in the centerpiece graphic of the Preamble, while more rigorous analytical results have been relegated to afterthoughts and footnotes.
>
> Studies that EPA Staff deemed to be of inferior quality have been presented as supportive without appropriate qualification.
>
> No apparent effort has been made to distinguish high-quality from lesser-quality studies with respect to the adequacy of their exposure assessments.

One might infer that failure to distinguish between strong and weak studies is motivated by the wish to avoid the exclusion of those positive findings that derive mainly from weaker studies.

5) The Preamble also misleads by its repeated statements that effects associated with coarse PM exposure were not affected by confounding by gaseous co-pollutants:

> "effect estimates ...are generally robust to confounding by gaseous co-pollutants" (*Fed Reg* 71:2660);
>
> "associations ... were largely unchanged in most cases when gaseous co-pollutants were included in the models" (*Fed Reg* 71:2657);

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[6] EPA has utilized such an approach for other risk assessments, such as in evaluating evidence of the carcinogenicity of trichloroethylene (e.g., "We divided the cohort studies into three tiers based on the specificity of the exposure information" (25)).

> "effect estimates ... are largely unchanged with the addition of gaseous co-pollutants to the models" (*Fed Reg* 71:2657).

But whether confounding is demonstrated depends on whether the correct co-pollutants have been included in the analytical model. Burnett et al. (1), for example, emphasized this concern: the apparent effects of particulates "could be completely explained by NO_2, a risk factor not as widely considered in North American locales as the other criteria pollutants." Similar conclusions were reached by Thurston et al. (12). Accordingly, the appropriate concern is not whether the effects of particulates are "generally robust", but whether potentially significant confounding has been properly evaluated. Such evaluations should consider "as many pollutants as possible" (12).

EPA has apparently not performed such evaluations. Instead, the Preamble relies on studies that incompletely evaluated possible confounding as evidence that such confounding is insignificant. However, the evidence provided by more rigorous studies indicates that confounding by gaseous co-pollutants can not be disregarded.

6) The limitations of the underlying evidence and the failure of EPA to adequately address and respond to those limitations are illustrated in the manner in which the Preamble argues that $PM_{10-2.5}$ is significantly associated with asthma. As described below, that argument is composed of hypothetical propositions and incorrect descriptions of cited studies.

a) The Preamble first proposes that because $PM_{10-2.5}$ might deposit in the tracheobronchial region, therefore it has the potential to aggravate asthma at the levels of exposure considered in the NAAQS. Following are examples of that proposition:

> "Deposition of particles to the tracheobronchial region is of particular concern with respect to aggravation of asthma" (*Fed* Reg 71:2654);
>
> "...has the potential to affect lung function and aggravate symptoms, particularly in asthmatics" (*Fed* Reg 71:2655);
>
> "The fractional deposition of elevated coarse particle concentrations is significant in the tracheobronchial region, which is particularly sensitive in asthmatic individuals." (*Fed* Reg 71:2661);
>
> "... the expectation that deposition of thoracic coarse particles in the respiratory system could aggravate effects in individuals with asthma" (*Fed* Reg 71:2668).

The hypothesis (or expectation) that $PM_{10-2.5}$ might aggravate asthma is not necessarily wrongheaded, but its repeated assertion provides neither support nor evidence that such a "potential" effect actually occurs.

b) The Preamble sometimes treats asthma (or "aggravation of asthma") as a distinct disease process, but more generally treats it as merely one of a number of more-or-less generic respiratory diseases.

The following statements, for example, suggest that EPA regards "asthma" as a distinct entity:

> "Evidence available in the last review suggested that aggravation of asthma ..." (*Fed Reg 71*:2656);
>
> "...limited epidemiologic evidence suggesting that aggravation of asthma..." (*Fed Reg 71*:2668);
>
> "The authors conclude that for acute asthma related responses..." (*Fed Reg* 71:2657).

In most places, however, the Preamble does not differentiate between asthma and a variety of acute respiratory diseases (e.g., respiratory infections, pneumonia) and chronic respiratory diseases (e.g., COPD). This is reflected by the following statements:

> "... respiratory morbidity effects, such as aggravation of asthma, increases in respiratory symptoms and respiratory infections..." (*Fed Reg 71*:2655);
>
> "...associations between short-term exposure to $PM_{10-2.5}$ with hospital admissions for respiratory diseases, including asthma, pneumonia and COPD..." (*Fed Reg 71*:2657);
>
> "...respiratory morbidity, such as increased respiratory symptoms and hospitalization for respiratory diseases such as asthma or COPD..." (*Fed Reg 71*:2661).

I suspect that the failure to distinguish asthma from those other acute and chronic diseases mainly reflects the paucity of published data specifically linking $PM_{10-2.5}$ and asthma.

c) The striking paucity of evidence linking $PM_{10-2.5}$ and asthma is made clear by the very few studies cited to support that association. Moreover, most of the cited studies provide less support than is implied in the Preamble.

The Preamble cites two studies [7], Hefflin et al. from southeast Washington and Gordian et al. from Anchorage, in which:

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[7] The Preamble actually cites "the last review" - 62 FR 38679 - in which these two studies were specifically identified.

> "...aggravation of asthma and respiratory infections and symptoms were associated with PM_{10} in areas where thoracic coarse particulate were a much greater fraction of PM_{10} than were fine particles" (*Fed Reg* 71:2657).

But, contrary to statements in the Preamble, Hefflin et al. (21) found no association between high-level exposure to PM_{10} and aggravation of asthma, even at 24-hour PM_{10} levels of 1035-1689 $\mu g/m^3$. To the contrary, those authors report:

> "... it is surprising that we not only found no significant association between PM_{10} and asthma, but we found relatively few emergency room admissions for asthma in a community that would be expected to have 4800 persons with asthma." (21)

The Gordian studies (17) suffer from potentially important flaws that limit its informational value. As discussed above, health effects were measured as doctors' visits, not episodes of illness, which may have led to overestimating the significance of associations. In addition, associations were noted for asthma and upper respiratory infections (URI) with a temporal relationship suggesting that onset of URI preceded the onset of asthma attacks [8]. Also, visits were not increased during the peak exposure days, when PM_{10} levels averaged 565 $\mu g/m^3$ and peak levels exceeded 3000 $\mu g/m^3$.

Notably, URI itself has been associated with asthma attacks in asthmatic children (22) and lower airway effects in normal children (23). Because of the apparent cause-and-effect relationship between URI and asthma attacks generally and the apparent correlation between URI and asthma visits in the Gordian study, it is not possible to determine whether those asthma visits reflected PM_{10} exposure vs. URI. In short, it is not possible to determine the extent to which the Gordian data might reflect the adverse effects of PM_{10}.

The Preamble also states that three "new US and Canadian epidemiologic studies" reported associations between short-term exposures to $PM_{10-2.5}$ with hospital admissions for "respiratory diseases, including asthma" (*Fed Reg* 71:2657). The three cited studies are from Toronto, Detroit and Seattle. However, the Preamble statement is incorrect and misleading.

> The Toronto study by Burnett et al. (1) did not find such an association. A multi-pollutant analysis found that any apparent association "was eliminated", with a relative risk of 1.007. It would be improper (and, perhaps, absurd) if EPA regarded relative risks of 1.007 as indicative of meaningful associations.

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[8] Doctors' visits for URI were most closely associated with same-day PM_{10} levels, while asthma visits were most closely associated with prior day PM_{10} levels. That suggests that URI preceded asthmatic symptoms. My personal experience (as an internist, emergency physician, parent and patient) is that visits for acute asthmatic attacks are more likely to occur shortly after the onset of symptoms, whereas URI visits occur after a longer delay, when symptoms and signs seem unusually persistent or severe. Thus, it seems likely that the onset of URI actually preceded asthma by several days.

> The cited Detroit study (16) was not a "new study", but a reanalysis of data from the older Lippmann et al. study (3). As discussed above, the Detroit study relied on particulate levels measured miles away in Windsor, Ontario and the HEI Health Review Committee concluded that data from the Detroit study were inconclusive.
>
> Likewise, the Seattle study (16) was not a "new study", but a reanalysis of Sheppard et al (15). As discussed above, exposure data were so lacking that 75% of coarse particulate data were "imputed". Also, the authors noted that wood burning was a "major contributor to PM", that vehicular exhaust was the second largest source of PM, and that the pollutant most closely associated with asthma was carbon monoxide, "an important environmental indicator of incomplete combustion, particularly from mobile sources" (15).
>
> Given the lack of measured coarse particulate data and the evidence that combustion-related fine particulate was an important pollutant in Seattle, there is essentially no basis to conclude that coarse PM in Seattle caused asthma-related hospitalizations.

The Preamble also mischaracterizes the findings of Schwartz and Neas (2) with respect to asthma. The Preamble states:

> "The authors conclude that for acute asthma related responses as well as daily mortality, fine particles are a stronger predictor of health response than are thoracic coarse particles." (*Fed Reg* 71:2657)

That statement implies that in addition to the large association seen with fine particles, Schwartz and Neas also found an association between coarse particles and asthma. That is not correct, as reflected by the authors' actual statements:

> "For lower respiratory symptoms, the association was stronger for all of the fine-particle measures than for CM [coarse particle mass] in single pollutant regressions. A model including both CM and $PM_{2.5}$ resulted in a substantial reduction in the effect of CM, with little evidence that the remaining effect was different from zero." (4)

EPA has incorrectly presented these negative findings as though Schwartz and Neas provided support for the Proposed Coarse PM NAAQS.

It is surprising to realize that the above studies reflect the totality of epidemiological data cited in the Preamble as support for the proposition that $PM_{10-2.5}$ aggravates asthma. These studies provide no such support, either individually or as a group.

d) There are other relevant studies that have been ignored in the Preamble discussion of asthma, perhaps because their findings showed no association of coarse particulate and asthma. Consider, for example, the three-year study by Rabinovitch et al. (24) that

specifically considered the effects of wintertime air pollutants on urban minority children at "highest risk for asthma morbidity". The children were students at a special school, operated at the National Jewish Hospital in Denver, which specifically enrolled children with chronic diseases including asthma. The school was located in a community where PM_{10} is dominated by coarse particulate; during the study period, coarse particulate on average comprised 61.2% of PM_{10}.

For two years, exposure data (including PM_{10} and $PM_{2.5}$) were obtained from EPA monitors located 100 meters from the school. During the third year, particulate data were obtained from a community monitoring station located 2.8 miles from the school. Children were monitored for asthma symptoms, asthma exacerbations, twice-daily FEV_1 and peak flows, use of asthma medications, and URI events. School activities were not modified in response to pollution alerts "so as not to bias any potential pollution effects".

Associations between air pollutants and asthma outcomes were found in simple models, but not in complex modeling that included all pollutants and time-dependent covariates such as URI events. Using the more complex model, no significant associations were observed between pulmonary function and PM_{10}. Asthma symptoms were significantly associated with ozone levels, but not PM_{10} and no significant associations were noted between asthma exacerbations and PM_{10}. By contrast, URI symptoms were strongly associated with decreased pulmonary function, increased medication usage, asthma symptoms, and asthma exacerbations.

These findings suggest that exposure to coarse particulate does not provoke asthma symptoms, does not adversely impact pulmonary function and does not induce asthma attacks. The strong associations seen between URI, pulmonary function and asthma lend support to the view that the results of the Gordian studies reflect URI events, rather than coarse particulate exposures.

e) In summary, it should be clear from the very few, very limited, and uncertain studies cited in the Preamble that there is no sound basis for concluding that coarse particulates aggravate asthma or provokes asthma symptoms, even at exposure levels considerably higher than those considered in the Proposed Coarse PM NAAQS. EPA arguments in favor of that association are composed of hypothetical propositions and incorrect or incomplete descriptions of the cited studies.

Conclusion

There is significant paucity of scientific support for the Proposed Coarse PM NAAQS and the scientific studies cited by EPA in support of the NAAQS suffer from significant methodological limitations.

Although EPA repeatedly acknowledges that the database suffers such limitations, it persists in presenting the accumulated data as sufficient to justify the Proposed NAAQS. But in addition to those acknowledged by the Agency, a detailed review of the cited

studies reveals numerous deficiencies that EPA has either not recognized or chosen to ignore.

Unlike many other EPA risk assessments that thoughtfully sorted strong from weak studies, emphasizing evidence from the former and discounting that from the latter, EPA in this case seems unwilling to discard any "finding" that might somehow be construed as supporting its NAAQS. That leads to important inadequacies in the justification and support of its proposed policy.

The majority of findings presented as supporting evidence derive from the methodologically weakest studies, while the methodologically most robust studies yield essentially no support. EPA relies on the least rigorous of analytical approaches (e.g., single pollutant models vs. multi-pollutant models), minimizes or ignores potential confounding (e.g., URI events inducing asthma attacks, gaseous co-pollutants) and, as discussed above, by misrepresenting study findings.

A detailed, balanced reading of the evidence indicates no basis to justify regulating of $PM_{10-2.5}$, only arguments and hypotheses that mainly reflect biological plausibility rather than empirical findings. The general lack of evidence persists even at exposure levels substantially higher than those considered health relevant in the Proposed Coarse PM NAAQS.

I find insufficient scientific justification for the adoption of the Proposed Coarse PM NAAQS.

Yours truly,

Jonathan Borak, MD, DABT, FACP, FACOEM

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The Honorable James M. Inhofe, Chairman
Senate Committee on Environment and Public Works
410 Senate Dirksen Office Building
Washington D.C. 20510

July 17, 2006

Dear Senator Inhofe:

We are pleased that you have convened a hearing to shed additional light on the scientific basis for revising the particulate matter standards. As researchers who have studied the health impacts of coarse particles ($PM_{10-2.5}$) in California's Coachella Valley, a desert resort area including Palm Springs, we wished to offer a few comments on the toxicity of coarse particles.

First, from a toxicological standpoint, there is extensive evidence that coarse particles elicit greater inflammatory responses in the lung in experimental settings than fine particles. Some of this inflammation is due to endotoxin, a chemical of microbial origin that is found in soils everywhere, and some is due to other constituents, including metals. Lung inflammation is considered one of the important mechanisms of particle toxicity.

Second, multiple studies examining effects of both fine and coarse particles on respiratory hospitalizations in the U.S. and Canada indicate a coarse particle effect as strong or stronger than that observed for fine particles.

Third, epidemiological studies of changes in daily mortality in relation to fluctuations in outdoor particle levels show associations with both fine and coarse particles. In most areas, the fine particle effect is stronger; however, in arid regions such as the Coachella Valley (a nonurban area), the associations tend to be greater for coarse particles.

Fourth, epidemiological studies have shown associations of coarse particles with cardiovascular hospitalizations (Detroit, Toronto) and cardiac deaths (Phoenix, Coachella Valley, and Mexico City).

Finally, there are few studies examining comparative effects of urban versus nonurban coarse particles. However, the data that do exist indicate that nonurban particles have the potential to cause adverse health effects among humans, including effects on heart function and cardiovascular mortality (see attached article). While there appear to be no published toxicology data on urban versus nonurban particles in the U.S., some recent research in Europe found that rural coarse particles were at least as pro-inflammatory as urban coarse particles, and both types of coarse particles were more toxic than urban fine particles. While there are relatively few studies on the health impacts of nonurban coarse particles, the limited published evidence indicates that these particles are not innocuous and may cause significant adverse effects on human health.

The Honorable James M. Inhofe
July 17, 2006

Nonurban particles are not just inert dust, but contain metals and harmful microbial products (including endotoxin), as well as other contaminants deposited from human activities, such as burning crop residues, applying pesticides and herbicides, and driving tens of thousands of vehicles/day on major highways through otherwise rural areas. Endotoxin, which is ubiquitous in rural soils, is associated with worsening of asthma as well as lung inflammation. In looking at the big picture, there is no compelling scientific justification for excluding nonurban particles from the scope of a national air quality standard intended to protect human health.

The opinions in this letter are our own and do not represent the official views of any of the institutions we are affiliated with, specifically the California Environmental Protection Agency (Cal/EPA) and the University of California, San Francisco. However, one of the enclosed attachments consists of the Cal/EPA comments submitted to the US EPA regarding the latter's proposed Particulate Matter National Ambient Air Quality Standards. We hope that our letter and the enclosures are useful in your review.

Sincerely,

Bart Ostro, PhD, Chief
Air Pollution Epidemiology Section,
California Office of Environmental Health Hazard Assessment

Michael Lipsett, MD
Associate Clinical Professor
School of Medicine
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Cc: The Honorable James M. Jeffords, Ranking Member
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The Honorable George V. Voinovich, Chairman
Senate Committee on Environment and Public Works
Subcommittee on Clean Air, Climate Change and Nuclear Safety
524 Hart Senate Office Building
Washington, D.C. 20510

The Honorable Barbara Boxer, Member
Senate Committee on Environment and Public Works
112 Hart Senate Office Building
Washington, D.C. 20510

The Honorable Thomas R. Carper, Ranking Member

The Honorable James M. Inhofe
July 17, 2006

Senate Committee on Environment and Public Works
Subcommittee on Clean Air, Climate Change and Nuclear Safety
513 Hart Senate Office Building
Washington, D.C. 20510

CHAIR, BUDGET SUBCOMMITTEE
ON RESOURCES (NO. 3)
COMMITTEE MEMBER:
BUDGET
EDUCATION
TRANSPORTATION
WATER, PARKS AND WILDLIFE

Assembly
California Legislature

FRAN PAVLEY
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April 13, 2006

The Honorable Stephen L. Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania, Avenue, NW
Washington, DC 20460

Re: EPA Docket ID No. OAR-2001-0017

Dear Mr. Johnson:

We understand that the United States Environmental Protection Agency (US EPA) may reject the fine particle standard that was recommended by the independent Clean Air Act Scientific Advisory Committee and USEPA staff scientists, opting instead only to slightly lower the standard to levels which may not protect the health of our citizens. We are writing to urge you to reconsider this proposal, and to see to it that EPA will adopt the more rigorous standard recommended by scientific and health experts to protect the public.

Particle pollution is both dangerous and pervasive, posing a serious and costly public health problem. Particle pollution causes asthma attacks, heart attacks, strokes, and lung cancer and cuts short the lives of tens of thousands of Americans every year. California suffers from some of the worst particle pollution in the nation.

Strong national standards are crucial to fighting this proven killer. Since particle pollution can travel long distances from its source, states cannot solve this problem on their own.

The Clean Air Act requires that air quality standards be based solely on the health effects of air pollutants. Under the Clean Air Act, air quality standards must be set at levels that protect public health, including the health of sensitive populations, with an adequate margin of safety. The current proposal, which largely maintains the status quo for fine particle pollution, is inadequate to protect public health. EPA's own analysis shows that the proposed fine particle standards will leave millions of Americans unprotected.

US EPA should issue strong final standards for fine particle pollution that protect public health and comply with the law. The administration should adopt an annual standard no higher than 12 micrograms per cubic meter and a daily standard no higher than 25 micrograms per cubic meter when it finalizes the standards in September.

In addition, US EPA should issue standards and monitoring requirements for coarse particle pollution that protect all Americans. The administration's proposal to eliminate pollution monitoring in small and mid-sized communities and to categorically exempt agriculture and mining from control requirements is unprecedented and unjustified.

We would appreciate a response to this request. Thank you very much for your consideration.

Sincerely,

Assembly Member Fran Pavley
41st Assembly District
California State Assembly
Chair, Legislative Environmental Caucus

Senator Sheila James Kuehl
23rd Senate District
California State Senate
Co-Chair, Legislative Environmental Caucus

April 15, 2006

The Honorable Stephen Johnson
Administrator
U.S. Environmental Protection Agency
1200 Pennsylvania Ave. NW
Washington, DC 20460

Re: National Ambient Air Quality Standards for Particulate Matter
71 Fed. Reg. 2620; Docket ID No. EPA-HQ-OAR-2001-0017; and Proposed Monitoring Requirements for Coarse Particles 71 Fed. Reg. 2710; Docket ID No. EPA-HQ-OAR-2004-0018

Dear Administrator Johnson:

We are writing to express our grave concern that the proposed revisions to the National Ambient Air Quality Standards for fine and coarse particulate matter (PM) are insufficient to protect public health and welfare as required by the Clean Air Act.

Under the Clean Air Act, primary standards must protect public health, including the health of sensitive populations, with an adequate margin of safety. Secondary standards must protect public welfare, including important adverse effects such as visibility impairment and damage to materials and crops. The proposed standards fail on both counts.

The adverse health effects of particulate matter are serious and have been well documented in EPA's Criteria Document and Staff Paper. The thousands of studies published over the last nine years make a much stronger case for the regulation of fine particles than in 1997, and indicate that the current standards must be lowered to protect public health.

Community health studies have consistently demonstrated associations between daily increases in fine particles and decreased lung function, exacerbation of asthma, more frequent emergency department visits, increased risk of heart attacks and strokes, additional hospital admissions, and increased number of daily deaths.

These effects have been demonstrated in cities where the daily concentrations of $PM_{2.5}$ are well below the current standard and rarely reach the level of the proposed 24-hour standard. Furthermore, the form of the proposed standard excludes too many of the most polluted days from compliance determinations.

Long term exposures to fine particles are implicated in premature death from heart disease, lung disease, and lung cancer. Lives may be shortened by one to three years. EPA's risk assessment demonstrates that thousands of premature deaths attributable to

particulate air pollution are occurring each year under the current standard, and that the proposed standards would do little to reduce this toll.

Building on earlier work, the largest ever epidemiological study of the effects of $PM_{2.5}$ in 204 U.S. counties was published in the *Journal of the American Medical Association* in March 2006.[1] This study showed clearly that the proposed standards for $PM_{2.5}$ fail to protect public health as required by the Clean Air Act. In this study, the average of the county mean annual values was 13.4 $\mu g/m^3$—well below the proposed standard of 15 $\mu g/m^3$. At levels below what EPA proposes as an annual standard, the findings showed cardiovascular and respiratory hospital admissions for the elderly increasing as concentrations $PM_{2.5}$ increased. Significant associations with excess cardiac and respiratory admissions persisted even after excluding all days above 35 $\mu g/m^3$ (the level of the proposed daily standard) from the study.[2] Even where $PM_{2.5}$ concentrations met *both* the proposed annual and 24-hour standards, serious health effects occurred.

Furthermore, a follow-up to the Harvard Six Cities Study published in March 2006 documented the life-saving benefits from reduced particulate levels. That study found that an average of three percent fewer people died for every reduction of one $\mu g/m^3$ in the annual average levels of $PM_{2.5}$.[3]

According to EPA's Children's Health Protection Advisory Committee, the proposed annual $PM_{2.5}$ standard does not provide the required adequate margin of safety to protect infants and children. The Committee concluded that the proposed daily $PM_{2.5}$ standard must also be revised downward to protect public health.[4]

The Clean Air Scientific Advisory Committee to the EPA has indicated that $PM_{2.5}$ causes adverse health effects including premature death at annual concentrations below the current standard, and has reiterated its recommendations for lowering the annual standard.[5]

Coarse particles are associated with increased hospitalization for respiratory infections in children, decreased lung function, increased hospital admissions for heart disease, increased hospital admissions for respiratory disease in the elderly and increased risk of

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[1] Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, Samet JM. Fine Particulate Air Pollution and Hospital Admission for Cardiovascular and Respiratory Diseases. *JAMA* 2006; 10:1127-1134.

[2] Letter from Francesca Dominici to U.S. EPA, March 23, 2006. Docket ID No. EPA-HQ-OAR-2001-0017-0988.

[3] Laden F, Schwartz J, Speizer FE, Dockery DW. Reduction in Fine Particulate Air Pollution and Mortality: Extended Follow-up of the Harvard Six Cities Study. *Am J Respir Crit Care Med* 2006; 173: 667-672.

[4] Letter from Melanie A. Marty, Ph.D., Chair, EPA Children's Health Protection Advisory Committee, to Stephen L. Johnson, Administrator, U.S. Environmental Protection Agency, RE: Proposed NAAQS for Particulate Matter, March 3, 2006.

[5] Dr. Rogene Henderson, Chair, Clean Air Scientific Advisory Committee letter to Stephen L. Johnson, Administrator, U.S. Environmental Protection Agency, March 21, 2006, Subject: Clean Air Scientific Advisory Committee Recommendations Concerning the Proposed National Ambient Air Quality Standards for Particulate Matter, EPA-CASAC-LTR-06-002.

premature death. EPA proposes a daily coarse particle standard that would be higher than levels where serious health effects have been reported in the studies EPA reviewed. EPA would enforce the standard only in urban areas with populations above 100,000, and exempt mining and agricultural sources of particles. EPA must set a coarse particle standard that applies nationally and without exemptions, to protect the health of all Americans as the Clean Air Act requires. Furthermore, EPA must not revoke the PM_{10} standard in any area of the country without providing protection against backsliding.

The Children's Health Protection Advisory Committee has recommended that the level of the coarse particle standard be lowered, that standards apply nationwide, with monitoring in both urban and rural areas, and that the exemption for agriculture and mining be withdrawn. The Clean Air Scientific Advisory Committee has also opposed exempting specific industries and recommended establishment of a national coarse particle monitoring program in urban and rural areas.

Our organizations strongly support lowering both the annual average and the 24-hour fine particle standard, while tightening the way compliance with the standards is measured. We urge you to adopt protective coarse particle standards that will apply nationwide, with monitoring in both urban and rural areas. We oppose the special exemptions for agribusiness and mining.

In addition, we believe that EPA must establish secondary standards for fine particles that protect against deterioration of visibility caused by fine particle pollution, as recommended by the Clean Air Scientific Advisory Committee, and set secondary standards for coarse particles that apply nationwide to protect against the ecosystem damage and visibility degradation they cause.

Specifically, we favor:

- an annual average $PM_{2.5}$ standard of 12 $\mu g/m^3$ or below, with elimination of the spatial averaging loophole;
- a 24-hour $PM_{2.5}$ standard of 25 $\mu g/m^3$, 99^{th} percentile;
- a 24-hour $PM_{10-2.5}$ standard of 25-30 $\mu g/m^3$, 99^{th} percentile, to apply nationally.
- Application of the course particle standard across the country, with monitoring in rural areas and elimination of the special exemption for the mining and agriculture industries;
- a $PM_{2.5}$ secondary standard of 20 $\mu g/m^3$ or below, based upon a rolling 4-hour average; and
- a secondary standard for coarse PM that protects ecosystems and scenic vistas across the country.

The decision over the air quality standards for fine and coarse particulate matter is the most far-reaching environmental health decision you will make this year. We urge you to strengthen the proposed standards, consistent with the law and the science.

Sincerely,

National Organizations

Center for Science in the Public Interest
Clean Air Task Force
Clean Air Watch
Clear the Air
Environmental Integrity Project
Friends of the Earth
Greenpeace USA
Izaak Walton League of America
League of Conservation Voters
League of Women Voters of the United States
National Audubon Society
National Environmental Trust
Public Citizen
Science and Environmental Health Network
Sierra Club
Union of Concerned Scientists
Unitarian Universalist Ministry for Earth
U.S. PIRG
Women's International League for Peace and Freedom

Regional, State, and Local Organizations

Alabama
Alabama Environmental Council

Alaska
Alaska Community Action on Toxics

Arizona
Arizona Public Interest Research Group
Grand Canyon Trust

California
Bay Area Clean Air Task Force
Bluewater Network
Center on Race, Poverty & the Environment
Coalition for Clean Air
Environment California
Fresno Metro Ministry
Global Community Monitor
Latino Issues Forum
Literacy for Environmental Justice
Long Beach Alliance for Children with Asthma

Our Children's Earth Foundation
Pacific Institute
Planning and Conservation League
Regional Asthma Management and Prevention (RAMP) Initiative, Oakland
Relational Culture Institute, Fresno
Steven and Michele Kirsch Foundation
The Regeneration Project/Interfaith Power & Light Campaign
Transportation Solutions Defense and Education Fund (TRANSDEF)
West Oakland Environmental Indicators Project

Colorado
Environment Colorado
Rocky Mountain Clean Air Action

Connecticut
Connecticut Coalition for Environmental Justice
Connecticut Public Interest Research Group

Florida
Big Bend Climate Action Team
Florida League of Conservation Voters
Florida Public Interest Research Group
Legal Environmental Assistance Foundation

Georgia
Georgia Conservancy
Georgia Public Interest Research Group
Mothers and Others for Clean Air, Atlanta

Illinois
American Bottom Conservancy
Citizens Against Ruining the Environment - C.A.R.E.
Illinois Public Interest Research Group
Lake County Audubon Society
Lake County Conservation Alliance
Mobile C.A.R.E. Foundation, Chicago
Stand Up/Save Lives Campaign

Indiana
Airaware
Indiana Public Interest Research Group
Valley Watch, Inc.

Iowa
Iowa Environmental Council
Iowa Public Interest Research Group

Kentucky
Kentucky Resources Council, Inc.

Louisiana
Alliance for Affordable Energy

Maine
Environment Maine
Friends of Acadia
Maine Breast Cancer Coalition
Maine Cardiovascular Health Council
Maine Public Health Association
Natural Resources Council of Maine
The Chewonki Foundation

Maryland
Chesapeake Bay Foundation
Maryland Public Interest Research Group

Massachusetts
Environmental League of Massachusetts
HealthLink
Massachusetts Public Interest Research Group
New England Clean Water Action
Sciencecorps

Michigan
HEAT - Hamtramck Environmental Action Team
Lone Tree Council
Michigan Coalition on the Environment and Jewish Life
Michigan Environmental Council
Michigan Interfaith Power & Light
Michigan League of Conservation Voters
Public Interest Research Group in Michigan

Mississippi
Environmental Coalition of Mississippi
Mississippi 2020 Network Inc.

Missouri
Health & Environmental Justice
Missouri Public Interest Research Group

Minnesota

Minnesota Center for Environmental Advocacy
Minnesotans for an Energy-Efficient Economy (ME3)

Montana
Montana Environmental Information Center
Montana Public Interest Research Group

New Hampshire
Aurum Foundation
C.L.E.A.R. Citizens Leading for Environmental Action and Responsibility
Citizens for a Sludge Free Land
Environmental Stewardship Committee of the ABC of Vermont and New Hampshire
New Hampshire Citizens for Sustainable Population
New Hampshire Global Warming Campaign
New Hampshire Pediatric Society
New Hampshire Public Interest Research Group

New Jersey
U.S. Environmental Watch, Elizabeth
New Jersey Public Interest Research Group

New Mexico
New Mexico Public Interest Research Group

New York
Atlantic States Legal Foundation, Inc.
Hudson River Sloop Clearwater, Inc.
Kids Against Pollution
New York Public Interest Research Group
Sustainable Energy Alliance of Long Island
West Harlem Environmental Action, Inc. (WE ACT)

North Carolina
Appalachian Voices
Blue Ridge Environmental Defense League
Clean Air Community Trust
Carolinas Clean Air Coalition
Environment North Carolina
North Carolina Conservation Network
Southern Alliance for Clean Energy
Southern Appalachian Biodiversity Project
Southern Environmental Law Center

Ohio
Environmental Health Watch
Green Environmental Coalition

Ohio Environmental Council
Ohio League of Conservation Voters
Ohio Public Interest Research Group
Western Lake Erie Waterkeeper

Oregon
Better Breathers Club, Medford
Northwest Environmental Advocates
Oregon Public Interest Research Group
Oregon State Public Interest Research Group
Rogue Valley Citizens for Clean Air

Pennsylvania
Army for a Clean Environment, Inc.
Center for the Celebration of Creation
Citizen Power
Citizens for Pennsylvania's Future
Clean Air Council, Philadelphia
Ecology Mission Group of Central Baptist Church
Group Against Smog and Pollution, Pittsburgh
PennEnvironment
Women's Health and Environmental Network

Rhode Island
Rhode Island Public Interest Research Group

South Dakota
Defenders of the Black Hills

Texas
Galveston-Houston Association for Smog Prevention (GHASP)
Environment Texas
Mothers for Clean Air, Houston

Utah
Great Salt Lake Keeper
Sevier Citizens for Clean Air and Water
Utah County Clean Air Coalition
Utah Population and Environment Coalition
Wasatch Clean Air Coalition

Vermont
Vermont Public Interest Research Group

Virginia
Greater Roanoke Valley Asthma and Air Quality Coalition

Piedmont Environmental Council

Washington
Washington Environmental Council
Washington Public Interest Research Group

Wisconsin
Clean Air Madison
Clean Wisconsin
Wisconsin Public Interest Research Group

Wyoming
Biodiversity Conservation Alliance
Western Watersheds Project
Wyoming Outdoor Council

United States Senate

WASHINGTON, DC 20510

April 7, 2006

The Honorable Conrad Burns
Subcommittee on Interior and Related Agencies
Committee on Appropriations
United States Senate
Washington, D.C. 20510

The Honorable Byron Dorgan
Subcommittee on Interior and Related Agencies
Committee on Appropriations
United States Senate
Washington, D.C. 20510

Dear Chairman Burns and Ranking Member Dorgan:

We respectfully request $220.3 million in Fiscal Year 2007 for the State and Local Air Quality Management Program – which is the amount appropriated in FY2006. The President's FY2007 budget reduces funding for this important program by $35.1 million.

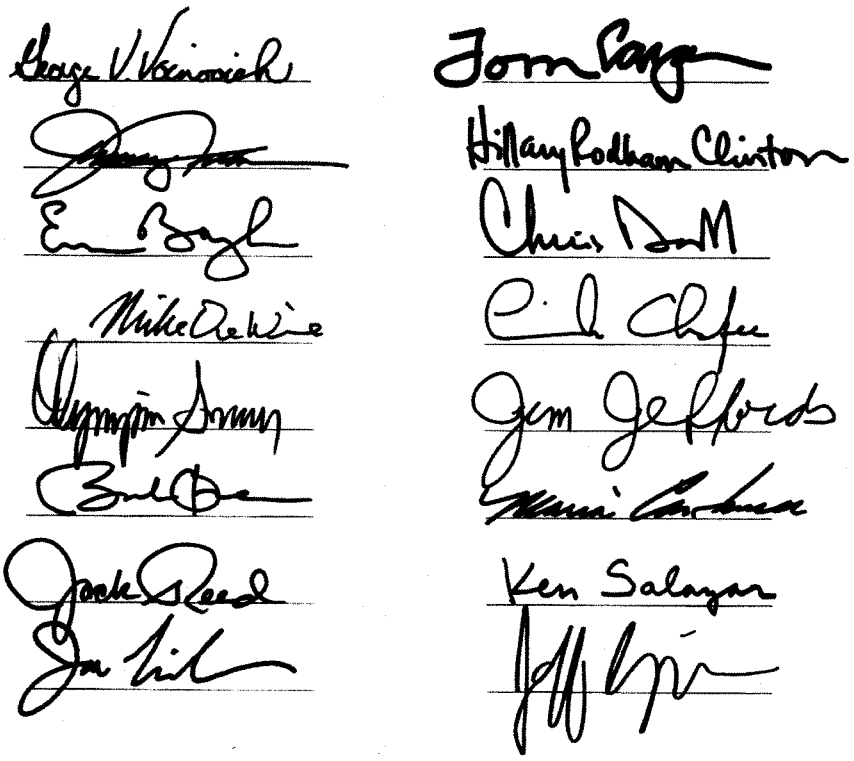
Established by the Clean Air Act, the State and Local Air Quality Management Program provides federal financial assistance in the form of grants to the 50 states, 4 territories, and approximately 60 local agencies to operate their air pollution control programs. The grants provide the resources to states and localities to perform basic air pollution control activities like monitoring air quality, developing and planning control options, permitting and inspecting sources, enforcing laws and regulations, and educating the public.

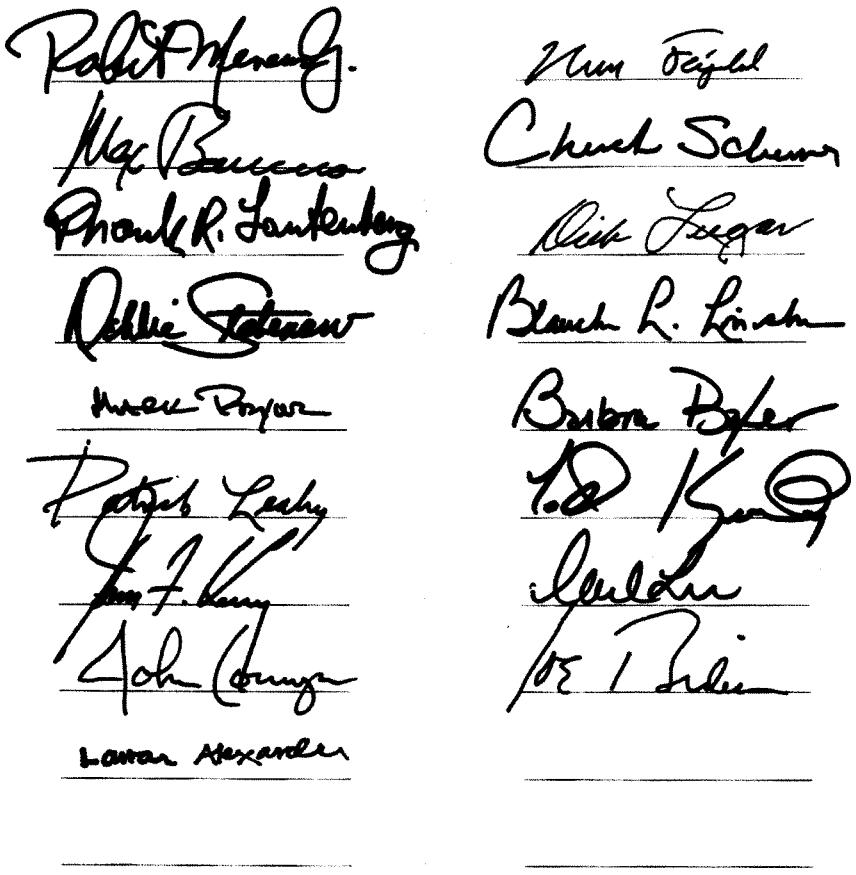
In particular, this grant funding helps support state and local air quality management efforts to implement the National Ambient Air Quality Standards. As you may know, the Environmental Protection Agency (EPA) has designated 495 counties across the nation as in nonattainment for the particulate matter and ozone air quality standards. Under the Clean Air Act, states must submit State Implementation Plans by 2007 and 2008 detailing how these areas will meet the standards by specified deadlines. Additionally, EPA has promulgated numerous other regulations that impose additional duties on state and local officials.

The President's FY2007 budget includes $185.2 million for the state and local grant program – which is a reduction of 16 percent or $35.1 million from the FY2006 appropriated level. This entails reductions of $15.6 million from the Section 105 air grants program and $2.5 million from regional planning organizations. Additionally, funding for the fine particulate monitoring program is cut by $17 million, with the remainder shifted from Section 103 authority to Section 105. This shift will require states and localities to provide matching funds.

Thank you for your consideration of this matter. We urge you to restore the funding for this important program to $220.3 million for FY2007 and not shift funds for monitoring from Section 103 authority to Section 105. Funding for this vital program should not be decreased at a time when the workload required of states and localities is increasing.

Sincerely,





United States Senate
WASHINGTON, DC 20510

April 28, 2006

The Honorable Conrad Burns
Subcommittee on Interior and Related Agencies
Committee on Appropriations
United States Senate
Washington, D.C. 20510

The Honorable Byron Dorgan
Subcommittee on Interior and Related Agencies
Committee on Appropriations
United States Senate
Washington, D.C. 20510

Dear Chairman Burns and Ranking Member Dorgan:

We are writing to join our colleagues who sent you the attached letter on the State and Local Air Quality Management Program. Funding for this vital program should not be decreased at a time when the workload required of states and localities is increasing.

We urge you to restore the funding for this important program to $220.3 million for Fiscal Year 2007.

Sincerely,

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